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*THE EFFECT OF THIOCTIC ACID ON THE QUANTUM EFFICIENCY OF THE HILL REACTION IN INTERMITTENT LIGHT**

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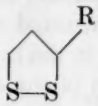
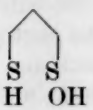
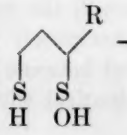
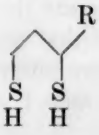
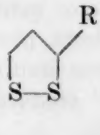
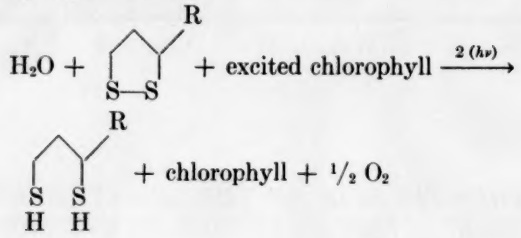
If thioctic acid is an intermediate in photosynthesis closely related to the primary photochemical act as proposed by Calvin and Barltrop,[1] it should be possible to realize experimental conditions under which the concentration of thioctic acid limits the quantum efficiency of photosynthesis as well as the closely related Hill reaction. Conditions under which the amounts of thioctic acid and oxidant, quinone, were simultaneously limiting factors in the Hill reaction in continuous light have been described by Bradley and Calvin.[2] A kinetic model consistent with this simultaneous limitation, the quantum conversion proposal of Calvin and Barltrop, and the photolysis experiments of Barltrop, Hayes, and Calvin[3] has been proposed by Bradley[4] (Model T). This consisted essentially of three steps: (1) a light-capturing step to produce a relatively long-lived electronic excitation; (2) a conversion step in which the "excitation" is converted into some "chemical" form consisting of an oxidizing and a reducing agent; and (3) the reduction of quinone by the reducing agent (in this model, the dithiol of thioctic acid) and the liberation of oxygen from the oxidizing agent. Two sets of more or less specific reactions which conform to these requirements are given below:

Step 1: Light + chlorophyll → excited chlorophyll

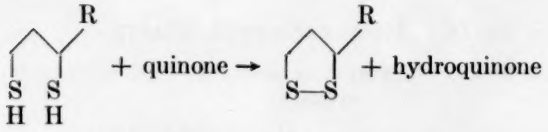
Step 2: H_2O +  + excited chlorophyll → chlorophyll + 

Step 3: 2  →  +  + ½ O_2 + H_2O

or Step 23 (in place of 2 + 3):



Step 4:



Step 5: Excited chlorophyll → chlorophyll (thermal decay)

Model T was shown by Bassham *et al.*[5] to be consistent with the available thermochemical data. The model is more general than that of Calvin[2] or Barltrop[3] or that of Levitt[6] as to the precise mechanism of energy transfer if step 2, and step 2 plus step 3, may be replaced by the less specific step 23.

It should be noted that, while the experiments of Bradley and Calvin are consistent with the quantum conversion process as conceived of by Calvin and Barltrop and by Levitt, the experiments could be carried out only under conditions in which the rate of oxygen evolution was limited by step 1 and/or step 4 and therefore provided no information as to the nature of step 2 (or step 23). Furthermore, these experiments did not establish unequivocally whether thioctic acid has *any* natural, in vivo function in the photochemical processes of photosynthesis.

The pioneering experiments of Emerson and Arnold,[7] in which they carried out photosynthesis in flashing light of high intensity with relatively long dark intervals between flashes, were designed to eliminate the rate limitation of both light capture (step 1) and dark, follow reactions (step 3 and further), the so-called "Blackman reactions" (e.g., steps 3 and 4). The limiting factor then supposedly becomes, in the photosynthetic unit models of Emerson and Arnold and of Wohl[8] (Model PU), the concentration of centers associated with several thousand chlorophyll molecules in the photosynthetic unit which may reduce CO_2 or, in the three-enzyme model of Franck and Herzfeld[9] (Model 3E), the concentration of the enzyme which reacts with the unstable photoproducts, the stabilizing enzyme B. In Model T the corresponding limiting chemical factor would become (in step 2) the concentration of thioctic acid in the disulfide form, provided that no other purely physical transformations which might become limiting were interposed between steps 1 and 2, i.e., the "excited chlorophyll" produced in step 1 is identical with the "excited chlorophyll" used in step 2 or 23.

An important assumption underlying such reasoning is that, when the rate of photon capture exceeds the possible rate of conversion into a suitable stable form (step 2), the excess quanta are lost through back reactions (e.g., step 5). This is based on the further assumption that the back reactions from the excited chlorophyll (step 5) occur rapidly with respect to the time needed to carry out the dark, follow reactions (e.g., steps 3 and 4), which seemed reasonable, since the lifetime

of the metastable state of chlorophyll in vitro has been shown by Livingston[10] to lie between 10^{-3} and 10^{-4} second, while the time for the completion of the dark reactions[7] has been found to lie between 10^{-1} and 10^{-2} second.

We have extended the original experiments of Bradley and Calvin, using the flashing-light technique of Emerson and Arnold in an attempt to eliminate the limitation of oxygen production by the dark chemical reactions, particularly step 4 (the reduction of quinone by thioctic dithiol), so that some information about the nature and possible thioctic acid requirement of step 2 (or step 23) might be obtained.

Experimental.—The experiments were carried out according to the basic protocol described previously[2] in an N_2 atmosphere to eliminate the necessity of a respiratory correction. Each vessel contained sufficient *Scenedesmus* to give an optical density of the extracted pigments from 1-ml. suspension in 10 ml. ethanol at the red maximum of chlorophyll a of 0.8 in a 1-cm. cell. This corresponds nearly to 10 mm^3 *Scenedesmus*/ml, or 20 mm^3 per vessel. Using the value of 8.3×10^4 as the molar extinction of chlorophyll a, this corresponds to 1.9×10^{-7} mole of chlorophyll a per vessel.[11]

Light flashes were produced by discharging capacitors through a GE FT422 high-pressure xenon blue-white flash tube (color temperature, 7,000° K.). The discharge was triggered by a variable high-voltage tickler trigger at rates adjustable from 0.5 to 100 flashes per second. The decay of light intensity was approximately exponential, with a time for half-decay of 160×10^{-6} second. The manometer vessels were so arranged with respect to the lamp that the intensity at 10 microfarads, 2,000 volts, and 10 flashes per second was 8×10^3 ergs/cm^2/sec, corresponding to 8×10^2 ergs/cm^2/flash. The flash rate was measured with a Techtronic scope at high flash rates and manually at rates below 5 per second. The intensity per flash was independent of flash rate in the region used, 5–20 cps. Yields of oxygen per flash were calculated from observed flash rates and the total yield over a 30–60-minute period of illumination.

Experimental Results.—A representative set of experimental values measured on aliquots of *Scenedesmus* from a single harvest is shown in Table 1. The probable experimental error (67 per cent standard deviation) in making such measurements was found to be 0.20×10^{-12} mole O_2/mm^3 *Scenedesmus* for aliquots from the same harvest, with considerably greater variation between harvests of algae on different days.

TABLE 1

DEPENDENCE OF YIELD PER FLASH UPON DARK INTERVAL BETWEEN FLASHES*
(Moles O_2 Evolved/Flash/Mm^3 *Scenedesmus* $\times 10^{12}$)

Dark Interval (Sec.)	Control	With Added 6T (0.27 Mg. or 7×10^{-4} *M*)	With 0.42 × Light	With 0.5 × Quinone
0.05	1.15	2.31	1.08	0.59
0.10	2.25	2.90	Omitted	1.26
0.20	2.96	2.72	1.62	2.70

* Temperature, 15.7° C.; voltage, 1,800 (20 cps.), 1,900 (10), 2,000 (5); *Scenedesmus*/vessel, 22 $mm.^3$; control quinone concentration, 0.0030 *M*; chlorophyll a/vessel, 1.9×10^{-7} mole; capacitance, 6.5 microfarads.

An experiment was carried out to compare the highest yields per flash obtainable with the amount of thioctic acid present in the plant. We are indebted to Dr. R. C. Fuller, who assayed the 6T for us by extracting aliquots of *Scenedesmus* with 6 *N*

HCl for one hour at 120° C., using the *S. faecalis* growth-assay method calibrated against synthetic *dl*-thioctic acid. Flashing-light experiments were carried out on aliquots from the same sample of *Scenedesmus*, the results appearing in Table 2.

TABLE 2

COMPARISON OF YIELD PER FLASH WITH IN VIVO THIOCTIC ACID CONCENTRATION*

(All Values in Moles/Mm³ *Scenedesmus* × 10^{12})

Oxygen per Flash (Duplicate)			6-Thioctic Acid (Triplicate)
Control	With 0.42 × Light	With 2.0 × Quinone	
2.94	1.33	2.32	3.8 ± 1 (mean dev.)

* Temperature, 15.7° C.; voltage, 2,100; *Scenedesmus*/vessel, 25 mm.²; control quinone concentration, 0.0030 *M*; Chlorophyll a/vessel, 1.9 × 10^{-7} mole; flash rate, 5 per second; capacitance, 10 microfarads; dark interval, 0.2 second.

Discussion.—From Table 1 we see that, when the interval between flashes is small, corresponding to continuous light, the oxygen evolution is limited by both quinone and 6T concentrations. These limitations are removed and replaced by light limitation when the dark period is lengthened, in agreement with the prediction based on Model T.

From Table 2 we see that the highest yields in molecules of oxygen per flash, when the dark time is sufficiently long, turn out to be very nearly equal to the total number of molecules of thioctic acid within the same amount of plant material. If we retain the assumption that each molecule of thioctic acid has time to convert quanta only once during the lifetime of the excitation produced by each flash, we arrive at the untenable result that not only does each quantum produce a molecule of oxygen but only one molecule of thioctic acid is involved in that production. We must therefore abandon the original assumption and permit each thioctic acid molecule to go through several complete cycles between successive flashes. Such multiple cycling of thioctic acid would be possible only if nonproductive decay of photoexcitation (e.g., step 5) were relatively slow. We can estimate the minimum lifetime of this excitation by calculating the minimum dark interval required for yield saturation for the four columns in Table 1: control, $2.96/2.25 \times 0.1 = 0.13$; added 6T, $2.96/2.31 \times 0.05 = 0.06$; $0.42 \times$ light, $1.62/1.08 \times 0.05 = 0.08$; $0.50 \times$ quinone, $2.96/1.25 \times 0.1 = 0.23$ second. Since the yield per flash is the same within probable error whether the recycling process requires 0.06 or 0.23 second for completion, it is evident that no appreciable nonproductive decay of photoexcitation occurs in 0.23 second.

This conclusion at first seems inconsistent with the finding of Livingston[10] that the lifetime of the photoexcited metastable state of chlorophyll in vitro lies between 10^{-3} and 10^{-4} second. However, Witt[12] has recently measured the lifetime of a photoexcited metastable absorption band at 5150 A in vivo in *Chlorella* which is of the order of 0.02 second. The fact that the long-lived photoexcited state of chlorophyll in vitro appears to have enhanced absorption at 5100 A led Witt to identify his excitation with that observed by Livingston. Although species differences might reasonably produce a lifetime as long as 0.06 second for Witt's metastable state in *Scenedesmus*, it is unlikely that his state can be identified with one that exhibits no appreciable decay in 0.23 second. We therefore conclude that in *Scenedesmus* also the metastable state showing enhanced absorption at 5150 A (Witt) decays in 0.02–0.06 second to a very long-lived state, >0.23 second.

The 0.23 second required for dark-time saturation with 0.50 × quinone (0.0015 *M*) is much longer than the time generally reported for photosynthesis and the Hill reaction at or near light saturation, i.e., 0.02 second for photosynthesis in *Chlorella* (Emerson and Arnold[7]) and 0.04 second for the Hill reaction (with quinone) in *Chlorella* (Clendenning and Ehrmantraut[11]). This long time results from quinone concentration limitation, as can be seen from the fact that the dark time for saturation is only 0.13 second at 0.003 *M* quinone. This inverse first-order dependence of minimum dark interval for flash saturation on quinone concentration does not extend far above 0.003 *M* because of the introduction of inhibitory side reactions by quinone. Our dark time for dark-interval saturation is therefore longer than previously observed because the *Scenedesmus* used are the first reported to be quinone-limited. We have grown *Scenedesmus*, for two years in continuous culture, which are quite reproducibly quinone-limited, although *Chlorella* grown under precisely the same environmental conditions are reproducibly non-quinone-limited. It is to be noted that, when quinone limitation is removed by the addition of thioctic acid (Table 1), the dark interval for saturation becomes 0.06 second, in approximate agreement with other values in the literature. The effects of quinone limitation closely parallel those of cyanide inhibition, studied by Weller and Franck,[13] in which the yield per flash is limited at short but not at long dark intervals and the minimum dark time for maximum yield per flash is much longer in the presence of cyanide, i.e., about 0.14 second. Weller and Franck, as well as Rieke and Gaffron,[14] interpret this as inhibition of the enzyme system which speeds the recovery of Catalyst B, and not inhibition of Catalyst B itself.

Speaking in more general terms, without reference to any specific model of the Hill system, it seems quite clear that low quinone concentrations limit oxygen production in continuous and rapid flashing light by limiting the rate at which the dark reactions are able to carry out the necessary oxidation reactions to remove the photoproducts and prepare the system for further photochemical reactions. The minimum dark time at 0.0015 *M* quinone is about 0.23 second; at 0.003 *M* quinone it has been shortened to 0.13 second, and it would be shortened still more by further additions of quinone if the higher quinone concentrations did not have some side effects which destroy the Hill reaction ability. The addition of relatively small amounts of thioctic acid (0.007 *M*) can shorten the minimum dark time to what appears to be its smallest value, i.e., 0.06 second. This thioctic acid effect is possible only with *Scenedesmus*, since it is only in *Scenedesmus* that the slower dark reactions leading to quinone reduction appear to be rate-limiting. A similar phenomenon can be achieved in other algae by partially slowing down some of the dark reactions with cyanide. We are thus led to the notion that, at least in *Scenedesmus*, it is possible to find conditions for the Hill reaction with quinone in which either one of two successive steps might be rate-limiting. The longer (later) one involves the reduction of quinone. When this rate limitation is removed either by raising the quinone concentration or by providing a more efficient hydrogen acceptor and carrier, such as thioctic acid, we then see the smallest time constant as rate-limiting (0.03–0.06 second).

This same time constant, or a corresponding one, has now been observed in three different types of experiments and is likely to be found in a fourth. These are, first, (*a*) the Emerson-Arnold dark times, first observed directly on photosynthetic

oxygen production and carbon dioxide reduction,[7] and (*b*) the corresponding dark times for the Hill reaction on whole *Chlorella*, reported by Clendenning and Ehrmantraut;[11] second, the decay time on whole *Chlorella* of some sort of excitation observed by Witt;[12] and, third, the minimum dark time for oxygen production in the Hill reaction in the presence of thioctic acid (present work). Fourth, an examination of the luminescence observations of Arnold and Strehler[15] indicates very clearly that the luminescence observed therein in all probability consists of at least two different processes. This is to be seen from the fact that (*a*) the apparent order of the luminescence decay changes with temperature, having an appearance of more nearly second order at 6° C. and more nearly first order at 25° C.; and (*b*) the temperature coefficients of the luminescence observed at ~0.1 second after the cessation of illumination vary greatly, being extremely small in the range of 5° and 10° C. and rising to somewhere between 10 and 20 kcal. in the range above 25° C. We thus suppose that this luminescence consists of at least two processes—a slower, high-temperature process, with a relatively high temperature coefficient (activation energy, approximately 15 kcal.), and a very fast process with a very small temperature coefficient. We suspect that this last-mentioned luminescence with the small temperature coefficient arises from the same species that has the 0.02-second decay time observed by Witt and whose transformation is responsible for the shortest minimum dark times measured by Emerson and Arnold (0.02–0.04 second) and by ourselves (0.06 second). The high-temperature luminescence with the long time constant would correspond to the decay of the long-lived excitation observed in our flashing-light experiments.

We are thus brought to the conclusion that the thioctic acid is not likely to be functioning prior to the 0.02–0.06-second transformation but that, after the 0.02–0.06-second transformation has taken place, the products formed by it have a very long intrinsic lifetime, as indicated by the possibility of finding conditions in which the minimum dark time is longer, as, for example, cyanide poisoning or the Hill reaction of *Scenedesmus* with quinone, and that the thioctic acid reacts with the products of this first transformation. A set of reactions which would correspond to such a proposal and which is, in effect, a modified Model T is as follows, and the relationships between the reactions are given in the following chart:

Step 1: Light + chlorophyll $\rightleftharpoons$ first-excited-state chlorophyll

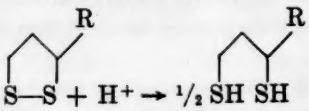
Step 2: First-excited-state chlorophyll $\rightarrow$ metastable-state chlorophyll

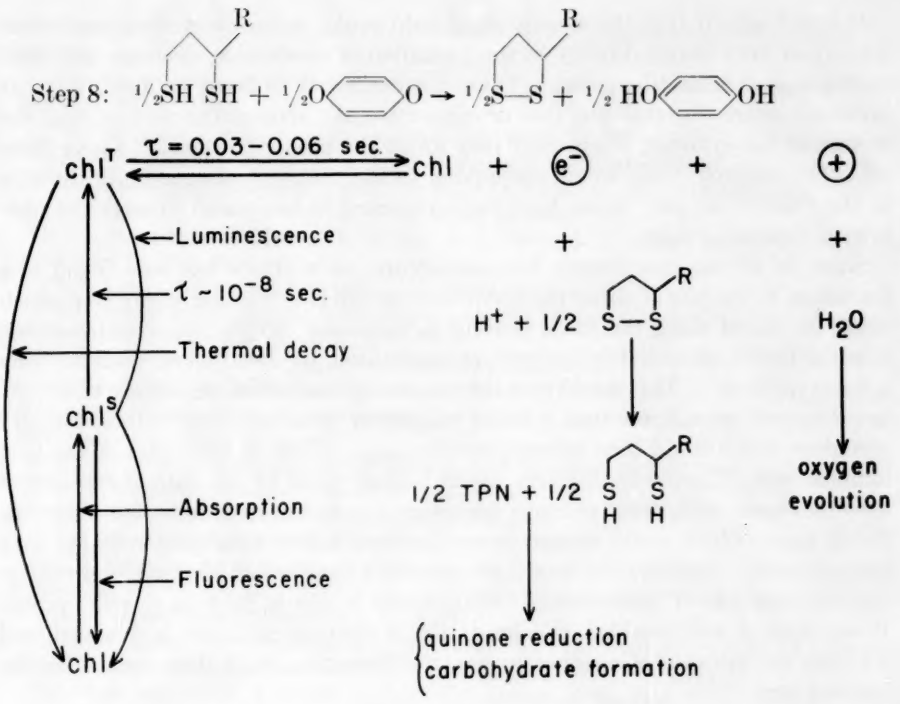
Step 3: Metastable chlorophyll $\rightarrow$ ground-state chlorophyll + heat

Step 4: Metastable chlorophyll $\rightarrow$ ground-state chlorophyll + luminescence

Step 5: Metastable chlorophyll $\rightleftharpoons$ ground-state chlorophyll + $\ominus$ + $\oplus$

Step 6: $\oplus + H_2O \rightarrow$ oxygen evolution

Step 7: $\ominus + \frac{1}{2}$  $+ H^+ \rightarrow \frac{1}{2}$



The primary question is, of course, first, the identity of the excitation which has a lifetime of 0.02–0.06 second and, second, the process by which it disappears and the products which are formed as a result of its disappearance. The long-lived (10^{-4} second) excited state of chlorophyll formed in solution (triplet state) has some indication of increased absorption in the range of 5100 A.[16] This has led Witt to identify his excitation, with its 0.02-second lifetime, with the triplet state as observed by Livingston in solution.

While it is possible that the hundred-fold increase in the lifetime of the metastable triplet state in vivo is associated with the rigidity with which the chlorophyll molecule is held in the grana, it seems more likely that the phenomenon is associated with the spectral shift observed in living systems which has been attributed to an interaction between chlorophyll molecules themselves as well as with other species. This leads directly to the concept of a quasi-crystalline lattice in which chlorophyll may actually exist in the plastid.[17] The recent elegant electron micrographs of Steinmann and Sjostrand[18] indicated clearly the presence of rather small, organized structures consisting of flat cylinders about 120 A thick and 3000–5000 A in diameter (and defined by lipid boundaries). The core of the cylinder is presumed to be filled with an aqueous phase, containing the porphyrin part of the chlorophyll molecule in some very definitely ordered array. The photochemical behavior of chlorophyll in such a situation would thus be on the border line between the behavior of molecules randomly oriented in true solution and the behavior of molecules completely oriented in a macrocrystal.[19]

It would appear that the absorption of light could, in such a system, lead either directly or very nearly directly to the formation of conduction electrons and their corresponding holes (ion pairs). These are conceived as being trapped, then, at suitable centers (for example, iron or copper atoms), arranged around or near the surface of the cylinder, where they may await the proper chemicals to take them off. The electron, then, would correspond to the "active hydrogen" and the hole to the "active oxygen" which have been presumed to be formed through the photolysis of water by light.

Since, in all the experiments done heretofore, no evidence has been found of a limitation in the rate of either photosynthesis or the Hill reaction of any step which could be placed along the chain leading to molecular oxygen, we would presume that the hole is immediately trapped, or neutralized, by donation of electrons from a water molecule. This would give the remaining conduction electrons a relatively long life, and we suppose that it is the transfer of these electrons to their primary acceptors which could then become rate-limiting. Thus at least part of the long luminescence observed by Strehler would be controlled by the rate of reaction of these electrons with their primary acceptors and successive reactions. Any oxidizing agent which could accept these electrons should thus obliterate the long luminescence. Further, the long minimum dark time which we have observed in the Hill reaction of *Scenedesmus* with quinone is due in part to this limitation. When thioctic acid is added, this limitation of electron oxidation is removed, and we then see the rate of conduction electron formation (0.02–0.06 second) as the limiting step.

Such a reaction, conceived as a process of diffusion of an "exciton"[20] through a quasi-crystalline lattice until "collision" with a suitable lattice imperfection leads to its conversion by ionization into a conduction electron and a positive hole, would be expected to have a small temperature coefficient and to be dependent upon a certain minimum-size particle.[18] The extremely high efficiency of thioctic acid as an electron acceptor in spite of its high reduction potential ($\sim$ 0.3 volt), is additional support for the suggestion that this is a natural function.

Summary.—The yield of oxygen per flash has been measured in *Scenedesmus* in flashing light. At dark intervals between flashes of 0.05 second the yield per flash is limited by the concentrations of both quinone and thioctic acid in the medium. When the dark interval is lengthened to 0.2 second, these limitations disappear and are replaced by light limitation. The yield per flash under these latter conditions is nearly equal to the total amount of thioctic acid in the plant on a molar basis, which is consistent with the proposal that thioctic acid is participating in the quantum conversion process in photosynthesis, provided that the lifetime for thermal decay of the photochemically active state (of chlorophyll) is at least of the order of 0.2 second in vivo. This, together with existing data, leads to the suggestion of two distinct physical stages in the conversion of the photon energy into chemical potential, only the second of which may involve thioctic acid.

We wish to express our appreciation to Dr. Wilson Powell and Mr. James Shand of the Radiation Laboratory for designing and constructing our flashing-light apparatus.

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*MAGNESIUM ANTAGONISM OF THE UNCOUPLING OF OXIDATIVE PHOSPHORYLATION BY IODO-THYRONINES**

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In parallel with the uncoupling of phosphorylation and respiration by nitro- and halophenols, uncoupling through the natural halophenols, thyroxine, triiodothyronine, and similar analogues has now been shown with increasing precision. Nevertheless, the fact that compounds of the nitrophenol type cannot replace thyroxine physiologically presents difficulty in the extension of such an uncoupling effect toward a generalized interpretation of thyroid hormone action. In the course of work on this problem, we became aware of a curious antagonism of magnesium against uncoupling with thyroxine. This is of particular interest inasmuch as this antagonism appears to be quite specific for the physiologically active iodothyro-

nines and is not obtained with monophenolic halo- and nitro-compounds. The first observation of an antagonism between thyroxine and magnesium was made by Lardy and his group[1] in studies on the inhibition of creatine kinase by thyroxine, an effect which first had been observed by Askonas.[2] Lardy and co-workers could show that thyroxine inhibition of magnesium-dependent creatine kinase could be overcome by increase of magnesium concentration with the simultaneous formation of an insoluble magnesium thyroxine compound. Although Lardy seems to be of the opinion that the inhibition by thyroxine of the kinase reaction is essentially due to the precipitation of the essential magnesium ions, a similar consideration seems not applicable in the case of the inhibition of aerobic phosphorylation, since, in contrast to the kinase, this antagonism exists to the same degree between triiodothyronine and magnesium, although the latter at similar levels does not form an insoluble magnesium compound and does not inhibit creatine kinase.[1] While this work was in progress, observations by Bain *et al.*,[3] rather similar to our own on an antagonism between magnesium and thyroxine, have appeared. We want to call attention, furthermore, to a similar antagonism between magnesium and aureomycin.[4]

MATERIALS AND METHODS

Mitochondria were prepared from freshly killed Syrian hamsters (Golden Nugget Hamstery, Maynard, Massachusetts) by differential centrifugation in 0.25 *M* sucrose and 0.15 *M* KCl, 0.01 *M* $NaHCO_3$,[5] and P/O ratios were measured as previously published.[5] The reaction mixtures for the measurement of oxidation and phosphorylation contained 120 μM glycylglycine, pH 7.4; 75 μM KCl; 40 μM phosphate; 30 μM β-hydroxybutyrate; 2 μM ATP; 1 μM DPN; and 0.02 μM cytochrome c. Other components were added to final concentrations as shown in the figures and tables. In the usual procedure 0.5 ml. of the mitochondrial suspension was added to the reaction mixture in the Warburg flask. If thyroxine or an iodinated derivative was to be added, it was dissolved in dilute NaOH and added *after* the mitochondria. In control vessels an equivalent amount of dilute NaOH was added. The side arm of the Warburg vessels contained 0.1 ml. of purified yeast hexokinase, step 3*a*,[6] and 0.1 ml. of 1.0 *M* glucose. The total volume was 3.0 ml. The flasks were equilibrated for 5–12 minutes at 28°; the hexokinase and glucose were then tipped from the side arm, and the oxygen uptake measured for 15–20 minutes. The reaction was stopped with 6 per cent TCA, and suitable aliquots were analyzed for content of inorganic phosphate. The P/O ratio was calculated from these determinations.

DL- and L-thyroxine, triiodothyronine, benzyloxy-3,5-diiodobenzoic acid, and *n*-butyl-3,5-diiodo-4-hydroxybenzoate were those used in previous publications from this laboratory. Triiodothyroacetic acid was kindly supplied by Dr. R. Pitt-Rivers. The ATP and DPN were obtained from Pabst Chemical Company and the cytochrome c from Sigma Chemical Company.

EXPERIMENTAL RESULTS

The basic phenomenon is well illustrated in Figures 1 and 2. There the effect is shown on the P/O ratio of an increasing magnesium concentration with constant concentration either of thyroxine (Fig. 1) or of triiodothyronine (Fig. 2). It is

important to note that in earlier experiments on uncoupling with thyroxine and similar compounds, before we became aware of the magnesium antagonism, routinely a magnesium concentration of 3.3 mM/liter was used (cf. Hoch and Lip-

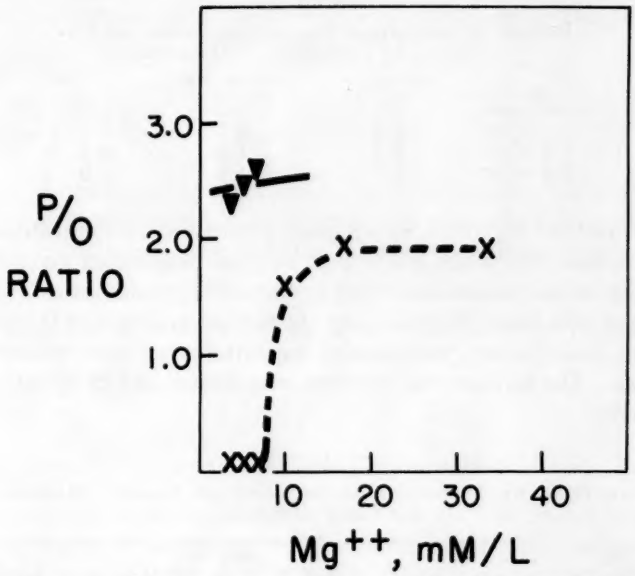

FIG. 1. Effect of magnesium on thyroxine uncoupling. —▼—▼—, control; X---X---, thyroxine, 10^{-4} M/liter.

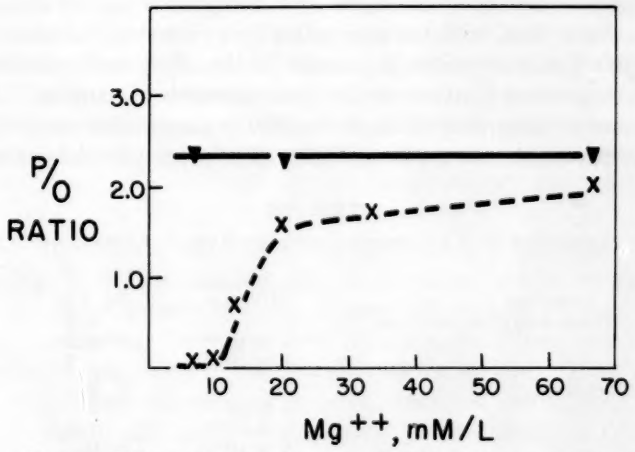

FIG. 2. Effect of magnesium on triiodothyronine uncoupling. —▼—▼—, control; X---X---, triiodothyronine, 2×10^{-4} M/liter.

mann,[5] p. 910). It may be seen in Figure 1 that up to the concentration of 6.6 mM/liter the system remains uncoupled but that recoupling appears at magnesium of 10 mM/liter. With triiodothyronine, a slightly higher concentration of magnesium is needed, and the effect is more gradual.

About 3–5 mM/liter of magnesium ion is optimal for phosphorylation. If the magnesium level is lowered to suboptimal concentration, phosphorylation becomes considerably more sensitive to thyroxine. This is illustrated in Table 1; at 0.7 mM

TABLE 1

EFFECT OF SUBOPTIMAL MAGNESIUM LEVELS ON UNCOUPLING BY THYROXINE (P/O RATIOS)

Mg^{++} (M/LITER)	THYROXINE (M/LITER) 0	5×10^{-6}	1×10^{-5}
7×10^{-4}	2.0	0.7	0.1
1.4×10^{-3}	2.3	1.0	0.2
2.8×10^{-3}	2.7	1.6	1.0

magnesium/liter the thyroxine shows good activity at concentrations as low as 5×10^{-6} M/liter, which are inactive at optimal magnesium concentration.

A generality of the magnesium effect for the iodothyronine series is further shown in experiments with triiodothyroacetate, the acetate analogue of thyroxine, a compound which recently has been studied by Pitt-Rivers[7] with regard to its thyroxine action. The antagonism between magnesium and triiodothyroacetate is shown in Table 2.

TABLE 2

UNCOUPLING BY TRIIODOTHYROACETIC ACID AT VARIOUS MAGNESIUM LEVELS (P/O RATIOS)

Mg^{++} (M/LITER)	TRIIODOTHYROACETIC ACID (M/LITER) 0	5×10^{-5}	1×10^{-4}	2×10^{-4}
6.7×10^{-3}	2.8	2.0	1.4	0.1
5×10^{-2}	2.7	2.3	1.8	0.4

Effect of Magnesium on Other Nitro- and Halophenols and on Aureomycin.—In Table 3 it is shown that, with the uncoupling by monocyclic iodophenol and nitrophenol, the effect of magnesium is opposite to the effect on the iodothyronines—that is, high magnesium increases rather than decreases uncoupling. Aureomycin, however, shows striking recoupling at the higher magnesium concentration.[4] In Table 4, an experiment with the benzyl ether of *p*-hydroxydiiodobenzoate is shown,

TABLE 3

EFFECT OF MAGNESIUM ON UNCOUPLING ACTION OF VARIOUS COMPOUNDS (P/O RATIOS)

SUBSTANCE	CONCENTRATION (M/LITER)	Mg^{++} (M/LITER) 6.7×10^{-3}	67×10^{-3}
N-butyl-3,5-diiodo-4-hydroxybenzoate	0	2.9	3.2
	5×10^{-5}	2.3	1.4
	1×10^{-4}	1.2	0.4
2,4-Dinitrophenol	0	3.2	2.5
	5×10^{-6}	2.1	0.9
	1×10^{-5}	1.0	0.1
	5×10^{-5}	0	0
Aureomycin	0	2.5	2.6
	1×10^{-4}	1.1	2.6
	2×10^{-4}	0	2.1

and with this compound there is a slight recoupling at higher magnesium concentration which appears significant, in particular, when compared to the increased uncoupling with high magnesium and nitro- and iodophenol. These results seem

to indicate that the antagonistic effect of magnesium is related to the phenol ether structure.

TABLE 4

UNCOUPLING BY BENZYL ETHER OF *p*-HYDROXYDIIODOBENZOATE AT TWO LEVELS OF MAGNESIUM (P/O) RATIOS)

Mg^{++} (M/LITER)	BENZYL ETHER (M/LITER) 0	1×10^{-6}	5×10^{-6}	1×10^{-5}	5×10^{-5}
3.3×10^{-3}	2.3	1.9	1.9	1.3	0
5×10^{-2}	2.5	2.4	1.8	1.8	0.2

Reversal of Uncoupling by Manganese Ion.—Manganese ion acts analogously to magnesium and has actually, mole per mole, a greater effect than the magnesium. This is shown in the experiment of Table 5. Here magnesium was present, 3.3×10^{-3} M/liter, but the addition of a little less than 10^{-3} M/liter of manganese ion causes nearly complete recoupling. A comparison with the concentration curve for magnesium, shown in Figure 1, indicates that a comparable increase in magnesium concentration would have had no effect. Other cations, including copper,[8] zinc,[9] and cobalt,[9] have been reported to form chelates with thyroxine, as shown by a change in the absorption spectrum. Cupric acetate and cobalt chloride under analogous conditions, however, did not antagonize thyroxine. Cupric acetate was used in equimolar concentration with thyroxine, while the cobalt salt was used in up to fourfold excess. In the cobalt experiment tris buffer was used instead of the glycylglycine to avoid chelation by the buffer. Experiments with zinc could not be carried out because of great toxicity of zinc at low concentrations.

TABLE 5

EFFECT OF MANGANESE ON THYROXINE UNCOUPLING (P/O RATIOS)

MANGANESE (M/LITER)*	THYROXINE (M/LITER) 0	2×10^{-4}
0	2.3	0.1
8×10^{-4}	2.9	2.0

* The medium contained, in addition, 3.3×10^{-3} M/liter Mg^{++}.

DISCUSSION

It has been known for a long time that magnesium is a component of the phosphorylation system in mitochondria; it is an essential part of the respiratory as well as the phosphate transfer system. It generally can be replaced in this type of reaction by manganese. Recent work by Lindberg, *et al.*,[10] may indicate some particular function of manganese in the phosphorylation system. With this in mind, the antagonism between magnesium or manganese and iodothyronines and analogues appears meaningful, and, in particular, the specificity of the magnesium effect for "natural" uncoupling agents appears worth noting.

It has been observed by various investigators that thyroxine is able to form chelates with certain metals, such as copper[8] and cobalt.[9] The fact that thyroxine forms a rather insoluble compound with magnesium has also been known for a long time.[11] However, triiodothyronine and the triiodothyroacetic acids seem not to form such magnesium complexes and are likewise antagonized by magnesium. This, at least, seems to rule out an effect due to a simple precipitation reaction. It should

be noted also that even at the low magnesium concentration, where thyroxine and the other thyronine derivatives are active, magnesium is present in excess of thyroxine. Furthermore, experiments with radioactive thyroxine reported previously[5] indicate that the actual concentration of thyroxine inside the mitochondria is very difficult to assess, since a considerable amount of the thyroxine added to the mitochondria suspension appears to be coprecipitated or adsorbed by the mitochondria.

Ample evidence is now available that the uncoupling effect of thyroxine with mitochondria depends on conditioning factors to render the mitochondria permeable to thyroxine.[5] The difficulties which have been encountered by various workers, including ourselves, in attempting to show uncoupling by thyroxine in an unambiguous manner appear to reflect in general the difficulty with which the thyroxine is able to reach the point of ultimate activity inside the mitochondria. Magnesium concentration may be a factor here. This may, however, be an oversimplification of the problem, because it appears more and more that the mitochondrial surface plays an important part in the phosphate transfer reaction.[12] It may well be that ATP actually does not enter the internal sphere of the mitochondria and that the pickup of ~P by the adenylic acid system is itself a surface reaction. It becomes rather difficult and probably meaningless to separate influences on the state of the mitochondrial surface from influences on phosphorylation if the phosphate transfer across the membrane to an external acceptor (ADP) may be an integral part of the phosphorylation complex. On the other hand, this would explain why processes concerning the surface structure of the mitochondria are, as we know, of the utmost importance for aerobic phosphorylation.

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The following abbreviations are used in this paper: ATP, adenosine triphosphate; DPN, diphosphopyridine nucleotide; DNP, 2,4-dinitrophenol; ADP, adenosine diphosphate; and TCA, trichloroacetic acid.

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*ALLELIC STRAINS OF NEUROSPORA LACKING TRYPTOPHAN SYNTHETASE: A PRELIMINARY IMMUNOCHEMICAL CHARACTERIZATION**

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The action of genetic material and its relation to enzyme biosynthesis has only in recent years received attention from an experimental standpoint. A variety of enzyme changes has been found to be associated with genetic alterations, including the formation of altered enzymes, the modifications in various quantitative aspects of enzyme formation, and the complete loss of ability to form specific enzymes (see review[1]). The first two categories have received some attention in previous investigations. The last category, which might be the most informative, has received scant attention in the past, largely because the lack of enzyme activity severely limits a direct experimental approach. However, the absence of detectable enzyme activity need not abolish all hope of examining such a condition. Even if the ability to form a specific enzyme were lost, there might remain the synthesis of proteins antigenically similar to that enzyme, and the presence of these could be investigated by immunochemical methods. This approach has recently been employed in studies of the β-galactosidase system of *Escherichia coli*[2] and of the tyrosinase of *Glomerella*.[3]

The present paper is a preliminary report on the application of immunochemical techniques to problems of gene action and of the control of tryptophan synthetase formation in *Neurospora crassa*. This enzyme was selected for study because a series of well-characterized alleles affecting its formation was available,[4, 5] as well as detailed information about the enzyme itself.[6, 7, 8]

Materials and Methods.—The various tryptophan independent (T^+) and tryptophan requiring (T^-) strains of *N. crassa* used in this work are listed in Table 1.

TABLE 1

STRAINS OF *N. crassa* EMPLOYED IN THE IMMUNOLOGICAL EXPERIMENTS

Strain	Tryptophan Requirement
Wild type (5256 A)	Tryptophan independent (T^+)
td_1, td_2, td_3, td_6, td_7	Tryptophan requiring (T^-)
td_1su_2	Tryptophan requiring (T^-)
td_2su_2, td_3su_3, td_6su_6	Tryptophan independent (T^+)

The tryptophan requiring strains employed, td_1, td_2, td_3, td_6, and td_7,[4, 5] represent independent mutations affecting the ability to convert indole to tryptophan. These mutants specifically lack tryptophan synthetase.[4, 5, 7] Genetic tests have indicated that these strains represent mutations of the same locus or of closely linked interdependent genetic material.[4, 5, 9] The tryptophan independent T^+ strains employed were of two types: parental strains and suppressed mutant strains (td su). As previously reported,[4, 5] suppressor mutations restore to specific td mutants the ability to grow in the absence of tryptophan and to form detectable amounts of tryptophan synthetase.

T^+ cultures were grown with aeration in 20 liters of glucose (1 per cent) minimal medium[10] for 60–70 hours at 30° C.; T^- cultures were grown in the presence of a supplement of 3 gm. of DL-tryptophan. The preparation, purification, and method of assay of extracts have previously been described.[8] The tryptophan synthetase unit (TSU) employed is the amount of enzyme which will catalyze the conversion of 0.1 μ mole of indole to tryptophan in a 60-minute incubation at 37° in the presence of saturating concentrations of indole, serine, and pyridoxal phosphate. Protein was determined by the methods of Lowry[11] and of Robinson *et al.*[12]

For immunization purposes an eightfold purified preparation of tryptophan synthetase was used as antigen. Four rabbits received approximately the same total dose of enzyme, 150 tryptophan synthetase units in about 45 mg. of *N. crassa* protein. All the rabbits were bled before receiving enzyme and again 6 weeks after the first injection. Inactivation of tryptophan synthetase by normal serum does not occur during incubation periods up to 1 hour. Normal serum inactivation of tryptophan synthetase does become a problem, however, if long incubation periods are used. This problem can be overcome by the use of sera which have been heat-treated and fractionated by the ammonium sulfate method of Tiselius.[13] Thus, for incubation periods up to 1 hour, whole untreated sera could be used. For long incubation periods, fractionated, heat-treated (56° C. for 30 minutes) sera were employed.

Precipitin reactions were ordinarily carried out at 5° C. At various times aliquots were removed, centrifuged in the cold, and carefully decanted. Enzyme determinations were routinely made on the supernatant solutions and occasionally on the precipitates. Enzyme assays were also run on uncentrifuged samples.

Absorption experiments were performed in a similar manner; the absorbing antigen was allowed to react with the serum at 5° C. for several hours before the addition of active enzyme. The removal of antibody against the active enzyme by this procedure was considered evidence for the presence of cross-reacting antigens in the absorbing extract. Inhibition values given are based on differences in activity between control serum and antiserum.

Results.—The antitryptophan synthetase activity of whole and fractionated immune serum was determined by measuring the reduction in activity of tryptophan synthetase preparations incubated with control serum and antiserum. From the inhibition values shown in Table 2, it can be seen that immunization with tryptophan synthetase preparations effectively elicits the formation of antibodies against this enzyme.

TABLE 2

INHIBITION OF TRYPTOPHAN SYNTHETASE ACTIVITY BY ANTISERUM, AS A FUNCTION OF TIME

Time at 5° C. (Hours)	Per Cent Inhibition by Antiserum	Time at 5° C. (Hours)	Per Cent Inhibition by Antiserum
0.25	65	48	72
1	66	72	69
24	74	168	53

Two tubes were tested at each time interval, one containing nonimmune (control) serum, and the other, antitryptophan synthetase antiserum. To each tube were added 1.0 ml. of a 1:1 dilution of the appropriate fractionated serum in phosphate buffer (0.1 *M*, pH 7.8), 0.2 ml. enzyme (14 TSU), 0.04 ml. pyridoxal phosphate (40 μgm.), and 0.1 ml. glutathione (5×10^{-2} *M*). At the indicated times the tubes were centrifuged, and the supernatants tested for enzyme activity. Inhibition values are based on difference in activity between control serum and antiserum.

It was of interest to determine two characteristics of this enzyme–anti-enzyme reaction: first, the time course of the reaction and, second, whether the antiserum neutralized the enzyme's activity or merely precipitated the enzyme. From Table 2 it can be seen that the reaction, measured in terms of tryptophan synthetase inhibition, is complete in less than 1 hour. It may be seen from Table 3 that the antiserum neutralizes enzyme activity, since there is no difference in percentage inhibition of enzyme activity between mixtures tested before centrifugation and those tested after centrifugation. Furthermore, no enzyme activity could be detected in the precipitate.

TABLE 3

NEUTRALIZATION OF TRYPTOPHAN SYNTHETASE ACTIVITY BY ANTISERUM

	Per Cent Enzyme Activity Remaining in		
Antiserum	Uncentrifuged Sample	Supernatant Solution after Centrifugation	Precipitate
1	70	74	...
2	52	46	...
3	0	0	0

Determinations were carried out in nonimmune serum and antitryptophan synthetase antiserum, and activity values are based on the difference in activity between these two sera. Antiserum from three rabbits was tested. To each tube were added 1.0 ml. of a 1:1 dilution of the appropriate fractionated serum in phosphate buffer (0.1 *M*, pH 7.8), 0.8 ml. enzyme (14 TSU), 0.04 ml. pyridoxal phosphate (40 μgm.), and 0.1 ml. glutathione (5×10^{-3} *M*). Tubes were stored at 5° C. for 40 hours before assay.

With a constant amount of antiserum and increasing amounts of enzyme, it is possible to standardize a given immune serum in terms of its antitryptophan synthetase activity. Absorption experiments were then carried out with extracts of various mutants to test for the presence in them of proteins antigenically related to tryptophan synthetase.

Extracts of strains td_1 and td_2 were employed in the initial absorption experiments. Aliquots of these extracts were incubated with antiserum of known antitryptophan synthetase titer for 1 hour at 5° C., the mixture centrifuged, and the supernatant solutions tested for their antitryptophan synthetase activity. As can be seen from the data in Table 4, strain td_2 contains proteins sufficiently similar to

TABLE 4

ABSORPTION OF ANTITRYPTOPHAN SYNTHETASE ANTISERUM WITH CRUDE AND FRACTIONATED PREPARATIONS OF STRAINS td_1 AND td_2

Absorbing Antigen	Test Antigen	Per Cent Inhibition of Tryptophan Synthetase Activity by Absorbed Antiserum
None	Tryptophan synthetase	100
td_1—crude extract	Tryptophan synthetase	100
td_1—dialyzed extract	Tryptophan synthetase	100
td_1—fractionated extract	Tryptophan synthetase	100
td_2—crude extract	Tryptophan synthetase	10
td_2—dialyzed extract	Tryptophan synthetase	12
td_2—fractionated extract	Tryptophan synthetase	3

To each tube were added 0.5 ml. of the appropriate fractionated serum, 0.5 ml. absorbing antigen, 0.04 ml. pyridoxal phosphate (40 μgm.), and 0.05 ml. glutathione (2.5×10^{-3} *M*). Tubes were adjusted to equal volume with phosphate buffer (0.1 *M*, pH 7.8). Absorption was continued for 2 hours at 5° C. before the test antigen (0.05 ml. enzyme [7 TSU]) was added. After an additional 30 minutes at 5° C., the tubes were assayed for enzyme activity. Determinations were carried out in nonimmune serum and antitryptophan synthetase antiserum, and activity values are based on difference.

tryptophan synthetase to react with and remove antibody to the enzyme, thereby reducing the anti-enzyme activity of the serum. Furthermore, the same results

are obtained regardless of whether the antiserum-extract mixtures are centrifuged after absorption or not. This observation indicates that the antigenically similar material inactivates or blocks the activity-neutralizing site of the anti-enzyme.

Of particular interest is the finding that extracts of strain td_1 do not contain cross-reacting protein.

Extracts of td_2 were fractionated in a manner similar to that used for purifying tryptophan synthetase preparations, and the final fraction tested for cross-reacting material. It can be seen in Table 4 that the cross-reacting material in extracts of strain td_2 is similar to tryptophan synthetase in its behavior during fractionation. Similar treatment of td_1 extracts failed to yield a preparation containing cross-reacting material.

In view of the differences observed between extracts of strains td_1 and td_2, extracts of other td mutants were examined for antigens which would absorb antitryptophan synthetase antibodies. As may be seen from Table 5, strains td_3, td_6, and td_7 all form proteins which cross-react with antitryptophan synthetase.

TABLE 5

ADSORPTION OF ANTITRYPTOPHAN SYNTHETASE ANTISERUM WITH EXTRACTS OF VARIOUS td MUTANTS AND OF SEVERAL td MUTANTS CARRYING SPECIFIC SUPPRESSOR GENES

Absorbing Antigen	Test Antigen	Per Cent Inhibition of Tryptophan Synthetase Activity by Absorbed Antiserum
td_1su_2—crude extract (0 TSU)	Tryptophan synthetase	100
td_2su_2—crude extract (0.3 TSU)	Tryptophan synthetase	19
td_2su_2—dialyzed extract (0 TSU)	Tryptophan synthetase	0
td_2su_2—fractionated extract (0.4 TSU)	Tryptophan synthetase	4
td_6su_6—crude extract (1 TSU)	Tryptophan synthetase	38
td_6—crude extract (0 TSU)	Tryptophan synthetase	36
td_3su_3—crude extract (0.65 TSU)	Tryptophan synthetase	16
td_3—crude extract (0 TSU)	Tryptophan synthetase	5
td_7—crude extract (0 TSU)	Tryptophan synthetase	13

To each tube were added 0.5 ml. of the appropriate fractionated serum, 0.5 ml. absorbing antigen, 0.04 ml. pyridoxal phosphate (40 μgm.), and 0.1 ml. glutathione (5×10^{-3} *M*). Absorption was continued for 2 hours at 5° C. before the test antigen was added (0.2 ml. enzyme [9 TSU]). After an additional 30 minutes at 5° C., the tubes were assayed for enzyme activity. Determinations were carried out in nonimmune serum and antitrytophan synthetase antiserum, and activity values are based on difference.

Several experiments were carried out with extracts of strains carrying suppressor genes. As may be seen in Table 5, extracts of td_1su_2 (strain td_1 is unaffected by su_2) do not contain cross-reacting proteins. Thus td_1su_2 and td_1 are similar in this respect. It can also be seen in this table that extracts of td_2su_2 *do* remove antibodies to tryptophan synthetase. This is not unexpected, since strain td_2su_2 forms small amounts of tryptophan synthetase.[4] However, what is surprising is that td_2su_2 extracts remove antibody out of proportion to the amount of active tryptophan synthetase they contain. This would suggest that this strain forms the antigen or antigens characteristic of td_2 in addition to the normal enzyme. The results obtained with extracts of strains td_6su_6 and td_3su_3 appear to be similar to those found with td_2su_2. Thus it would appear that suppressed strains possessing suboptimal tryptophan synthetase levels continue to form another protein or proteins, antigenically related to tryptophan synthetase but having no tryptophan synthetase activity.

Discussion.—The various tryptophan requiring strains used in these investigations are noteworthy in that they possess a number of properties in common. All

these strains have an absolute requirement for tryptophan which cannot be satisfied by either indole or anthranilic acid; they have similar requirements for tryptophan; they are characterized by their complete lack of tryptophan synthetase activity; and they all appear to be allelic. In view of these similarities, it may be said that these strains represent mutations of the same or of closely linked interdependent genetic material at the same biochemical locus.[4, 5]

These same mutants, however, do show some very striking differences, particularly in their response to specific suppressor genes;[4, 5] a given suppressor restores tryptophan synthetase formation only in combination with a particular td allele.

The results of the present investigation further demonstrate that differences exist between these allelic strains. Some of the mutants form material closely related antigenically to tryptophan synthetase, but this property is not characteristic of all the alleles. Whether the antigens formed by the various strains differ is not as yet known. It may well be that we are dealing with differences other than the presence or absence of a single cross-reacting antigen. Hence these experiments further strengthen the conclusion that a locus controlling the formation of a specific enzyme can be exceedingly complex and subject to numerous changes. The elucidation of differences between alleles would thus appear to be dependent upon the number of criteria that are applied.

The fact that td_1su_2 does not form cross-reacting material demonstrates that it is the mutant locus and not the suppressor which controls the production of the antigen or antigens. Of particular interest in this regard is the observation that td_2su_2, td_3su_3, and td_6su_6 appear to form both cross-reacting material and tryptophan synthetase. This would suggest that suppressor genes do not completely overcome defects in tryptophan synthetase formation, a conclusion consistent with the fact that suppressed strains form considerably less tryptophan synthetase than the wild-type strain.

One additional point of interest is that all attempts to obtain a suppressor for strain td_1, the strain which does not form cross-reacting material, have been unsuccessful.[4, 5] Td_2 and all the alleles studied except td_7 form cross-reacting material and are suppressed by specific suppressor genes. The suppressibility of strain td_7 has not as yet been demonstrated. These data seem to suggest that the ability to form cross-reacting material and suppressibility may be related. The validity of this correlation must await further study, particularly in cases such as that of td_7.

What is the relationship of the cross-reacting antigen or antigens to tryptophan synthetase? Certainly it is not known at this time, although a great many possibilities exist. The cross-reacting material could be enzyme precursor, altered or incomplete forms of the enzyme, inactivated tryptophan synthetase, or a protein or proteins formed along the same biosynthetic pathway as tryptophan synthetase. Further study is necessary to answer the question.

In any event, the presence or absence of proteins antigenically related to tryptophan synthetase in different members of a group of very similar mutants does suggest that these differences may be a reflection of defects in specific and separate phases of the synthesis of tryptophan synthetase.

Summary.—Preliminary data have been presented on the immunochemical characterization of the tryptophan synthetase–antitryptophan synthetase reaction. Antibody against partially purified tryptophan synthetase preparations from *N. crassa* was found to neutralize enzyme activity. Extracts of several allelic tryptophan requiring mutants, which lack the enzyme tryptophan synthetase, were examined for the presence of proteins which cross-react with antitryptophan synthetase antibody. All but one of the mutants studied were found to possess such material. The cross-reacting material in the mutants behaved as did tryptophan synthetase during the course of purification. Mutants which carry specific suppressor genes, with the attendant formation of low levels of tryptophan synthetase activity, were found to form both active enzyme and cross-reacting material.

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*ON ABELIAN VARIETIES OVER FUNCTION FIELDS**

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In a recent paper[1] on Abelian varieties over function fields, we have shown that to every algebraic system of Abelian varieties, defined over a field K, can be assigned two invariants, called the *K-image* and the *K-trace*. Let $K(u)$ be a primary extension of K, and let A^* be an Abelian variety defined over $K(u)$; then the K-image A of A^* over $K(u)$ is an Abelian variety over K characterized by the existence of a rational homomorphism F (called the *canonical homomorphism*) of A^* onto

A, defined over $K(u)$, such that, if H is a rational homomorphism of A^* into an Abelian variety B, and if B is defined over a separably generated extension K_1 of K, independent with respect to $K(u)$ over K, and H is defined over $K_1(u)$, then H is the product of F and a rational homomorphism of A into B, defined over K_1. The K-trace A' of A^* over $K(u)$ is defined in case $K(u)$ is a regular extension of K, and is an Abelian variety over K characterized by the existence of a rational isomorphism F (called the *canonical isomorphism*) of A' into A^*, defined over $K(u)$, such that if H is a rational homomorphism of an Abelian variety B into A^*, and if B is defined over an extension K_1 of K, independent with respect to $K(u)$ over K, and H is defined over $K_1(u)$, then H is the product of F and a rational homomorphism of B into A', defined over K_1. We observe that, while the K-trace is defined only for the case of a regular extension $K(u)$ of K, it possesses as a compensation the stronger property that the field K_1 involved is any extension of K which is independent with respect to $K(u)$ over K, not necessarily a separably generated one. Furthermore, as we have shown in Paper AF (Theorem 8, Corollary), the K-trace has the following property:

Let $u_1, \ldots, u_m$ be independent generic specializations of u over K, and let $A_1^*, \ldots, A_m^*$ and $F_1, \ldots, F_m$ be the corresponding specializations of A^* and F, respectively; if y is a generic point of A' over $K(u_1, \ldots, u_m)$, then the correspondence $y \to (F_1(y), \ldots, F_m(y))$ defines a regular isomorphism of A' into the product variety $\prod_{i=1}^{m} A_i^*$, provided that m is taken sufficiently large.

As we have indicated at the end of AF, there naturally arises the question whether a similar result also holds for the K-image A of A^* over $K(u)$ in case $K(u)$ is a regular extension of K; that is, if $(x_1, \ldots, x_m)$ is a generic point of the product variety $\prod_{i=1}^{m} A_i^*$ over $K(u_1, \ldots, u_m)$, whether the correspondence $(x_1, \ldots, x_m) \to \sum_{i=1}^{m} F_i(x_i)$ defines a regular homomorphism of $\prod_{i=1}^{m} A_i^*$ onto A, provided that m is sufficiently large. The purpose of the present note is to show that the answer to this question is in the affirmative. This result is significant, for in many applications it is advantageous to be able to deal with a regular homomorphism, and we do not know whether the canonical homomorphism F is regular; furthermore, as we shall see later, it follows easily from this result that the field K_1 mentioned above can be any extension of K which is independent with respect to $K(u)$ over K, in case $K(u)$ is a regular extension of K, so that in this case also the K-image is independent of any extension of the ground field K.

We recall that an extension K_1 of K is said to be *primary* if every element in K_1 which is separably algebraic over K is contained in K. We note that, if $K_1, \ldots, K_s$ are independent primary extensions of K, then the compositum $K_1 \ldots K_s$ is also a primary extension of K. In fact, there exist subfields $K_1', \ldots, K_s'$ in $K_1, \ldots, K_s$, respectively, such that each K_i' is a regular extension of K and K_i is a purely inseparable extension of K_i'; since $K_1', \ldots, K_s'$ are independent extensions of K, the compositum $K_1' \ldots K_s'$ is also a regular extension of K, and it is clear that $K_1 \ldots K_s$ is a purely inseparable extension of $K_1' \ldots K_s'$.

THEOREM. *Let $K(u)$ be a regular extension of K, and let A^* be an Abelian variety defined over $K(u)$; let A be the K-image of A^* over $K(u)$, and let F be the canonical*

homomorphism of A^* onto A. Let $u_1, \ldots, u_m$ be independent generic specializations of u over K, and let $A_1^*, \ldots, A_m^*$ and $F_1, \ldots, F_m$ be the corresponding specializations of A^* and F, respectively; if $(x_1, \ldots, x_m)$ is a generic point of the product variety $\prod_{i=1}^{m} A_i^*$ over $K(u_1, \ldots, u_m)$, then the correspondence $(x_1, \ldots, x_m) \rightarrow \sum_{i=1}^{m} F_i(x_i)$ defines a regular homomorphism of $\prod_{i=1}^{m} A_i^*$ onto A, provided that m is taken sufficiently large.

Proof: For convenience, we set $K_m = K(u_1, \ldots, u_m)$ and $A_{(m)}^* = \prod_{i=1}^{m} A_i^*$, and we denote by $F_{(m)}$ the rational homomorphism of $A_{(m)}^*$ onto A defined by the correspondence $(x_1, \ldots, x_m) \rightarrow \sum_{i=1}^{m} F_i(x_i)$. We maintain that the kernel $X_{(m)}$ of $F_{(m)}$ is an Abelian subvariety in $A_{(m)}^*$. In fact, according to AF, Theorem 6, Corollary 2, for each $i = 1, \ldots, m$, $K_m(x_i)$ is a primary extension of $K_m(F_i(x_i))$; it follows, then, from a remark we made above that $K_m(x_1, \ldots, x_m)$ is a primary extension of $K_m(F_1(x_1), \ldots, F_m(x_m))$. Since $F_1(x_1), \ldots, F_m(x_m)$ are independent generic points of A over K_m, the field $K_m(F_1(x_1), \ldots, F_m(x_m))$ is a primary extension of $K_m\left(\sum_{i=1}^{m} F_i(x_i)\right)$; it follows, then, that $K_m(x_1, \ldots, x_m)$ is a primary extension of $K_m\left(\sum_{i=1}^{m} F_i(x_i)\right)$, which shows that $X_{(m)}$ is an Abelian subvariety in $A_{(m)}^*$. Since $X_{(m)}$ is evidently normally algebraic over K_m, it follows from AF, Theorem 2, that $X_{(m)}$ is defined over K_m. According to a result of ours on the quotient varieties,[2] the canonical homomorphism $R_{(m)}$ of $A_{(m)}^*$ onto its quotient variety $A_{(m)}^*(X_{(m)})$ relative to $X_{(m)}$ is separable, and there exists a rational isomorphism $T_{(m)}$ of $A_{(m)}^*(X_{(m)})$ onto A, defined over K_m, such that $F_{(m)} = T_{(m)}R_{(m)}$. We set $y_{(m)} = R_{(m)}(x_1, \ldots, x_m)$ and $z_{(m)} = F_{(m)}(x_1, \ldots, x_m)$, so that we have the relation $z_{(m)} = T_{(m)}(y_{(m)})$.

Next we observe that the degree $[K_m(y_{(m)}) : K_m(z_{(m)})]$ is a monotone nonincreasing function of the integer m. To show this, denote by X_m the kernel of F_m; according to AF, Theorem 6, X_m is an Abelian subvariety in A_m^*, and, according to Theorem 2 of the same paper, X_m is defined over $K(u_m)$; according to the result on quotient varieties cited above,[2] the canonical homomorphism R_m of A_m^* onto its quotient variety $A_m^*(X_m)$ relative to X_m is separable, and there exists a rational isomorphism T_m of $A_m^*(X_m)$ onto A, defined over $K(u_m)$, such that $F_m = T_m R_m$. We set $y_m = R_m(x_m)$ and $z_m = F_m(x_m)$, so that we have the relations $z_m = T_m(y_m)$ and $z_{(m)} = z_{(m-1)} + z_m$. Since the Abelian subvariety $X_{(m-1)} \times X_m$ in $A_{(m)}^*$ is defined over K_m and since it is evidently contained in $X_{(m)}$, it follows[2] that there is a rational homomorphism of $A_{(m-1)}^*(X_{(m-1)}) \times A_m^*(X_m)$ onto $A_{(m)}^*(X_{(m)})$, defined over K_m, and $(y_{(m-1)}, y_m)$ and $y_{(m)}$ are a pair of corresponding generic points under this homomorphism. We have, therefore, the relation $K_m(y_{(m-1)}, y_m) \supset K_m(y_{(m)})$ and hence the relation $K_m(y_{(m-1)}, y_m) \supset K_m(y_{(m)}, y_m) \supset K_m(z_{(m)}, y_m) = K_m(z_{(m-1)}, y_m)$; since $K_m(y_{(m)}, y_m)$ is (for $m > 1$) a regular extension of $K_m(y_{(m)})$, it follows that $[K_m(y_{(m)}) : K_m(z_{(m)})] = [K_m(y_{(m)}, y_m) : K_m(z_{(m)}, y_m)] \leq [K_m(y_{(m-1)}, y_m) : K_m(z_{(m-1)}, y_m)] = [K_m(y_{(m-1)}) : K_m(z_{(m-1)})]$. This proves our assertion. We observe also that the relation $K_m(y_{(m-1)}, y_m) \supset K_m(y_{(m-1)}, y_{(m)})$ implies the existence of a rational homo-

morphism G of $A_m^*(X_m)$ onto $A_{(m)}^*(X_{(m)})$, defined over $K_m(y_{(m-1)})$; according to AF, Theorem 2, Corollary 1, G is also defined over K_m. On the other hand, the relation $K_m(y_{(m-1)}, z_m) = K_m(y_{(m-1)}, z_{(m)})$ implies the existence of a regular isomorphism G_1 of A onto itself, defined over $K_m(y_{(m-1)})$, which carries z_m into $z_{(m)}$; and according to AF, Theorem 2, Corollary 1, G_1 is defined over K. Finally, the relations $T_{(m)}G(y_m) = z_{(m)} = G_1(z_m) = G_1T_m(y_m)$ show that $T_{(m)}G = G_1T_m$.

From now on we shall assume that the integer m has been taken so large that the order $[K_m(y_{(m)}):K_m(z_{(m)})]$ attains its minimum value, and we have to show that this minimum value is equal to 1, i.e., $T_{(m)}$ is a regular isomorphism.

Now let $u_1', \ldots, u_m'$ be independent generic specializations of u over K_m, and denote by $A_{(m)}^{*'}, X_{(m)}', F_{(m)}', R_{(m)}', T_{(m)}', y_{(m)}', z_{(m)}'$ the same elements as those without the prime superscript introduced above, with the field K_m now replaced by the field $K_m' = K(u_1', \ldots, u_m')$; similarly, we denote by $A_{(2m)}^*, X_{(2m)}, F_{(2m)}, R_{(2m)}, T_{(2m)}, y_{(2m)}, z_{(2m)}$ the corresponding elements defined with respect to the field $K_{2m} = K(u_1, \ldots, u_m, u_1', \ldots, u_m')$. It is clear that $z_{(2m)} = z_{(m)} + z_{(m)}'$. Since the Abelian subvariety $X_{(m)} \times X_{(m)}'$ in $A_{(2m)}^*$ is defined over K_{2m} and since it is evidently contained in $X_{(2m)}$, it follows[2] that there is a rational homomorphism of $A_{(m)}^*(X_{(m)}) \times A_{(m)}^{*'}(X_{(m)}')$ onto $A_{(2m)}^*(X_{(2m)})$, defined over K_{2m}, and $(y_{(m)}, y_{(m)}')$ and $y_{(2m)}$ are a pair of corresponding generic points under this homomorphism. We have, therefore, the relation $K_{2m}(y_{(m)}, y_{(m)}') \supset K_{2m}(y_{(2m)})$ and hence the relation $K_{2m}(y_{(m)}, y_{(m)}') \supset K_{2m}(y_{(2m)}, y_{(m)}') \supset K_{2m}(z_{(2m)}, y_{(m)}') = K_{2m}(z_{(m)}, y_{(m)}')$; furthermore, by our choice of the integer m, we have the relations $[K_{2m}(y_{(m)}, y_{(m)}'): K_{2m}(z_{(m)}, y_{(m)}')] = [K_{2m}(y_{(m)}): K_{2m}(z_{(m)})] = [K_{2m}(y_{(2m)}):K_{2m}(z_{(2m)})] = [K_{2m}(y_{(2m)}), y_{(m)}'):K_{2m}(z_{(2m)}, y_{(m)}')]$. This shows that $K_{2m}(y_{(m)}, y_{(m)}') = K_{2m}(y_{(2m)}, y_{(m)}')$, and hence there exists a regular isomorphism H of $A_{(m)}^*(X_{(m)})$ onto $A_{(2m)}^*(X_{(2m)})$ defined over $K_{2m}(y_{(m)}')$; according to AF, Theorem 2, Corollary 1, H is also defined over K_{2m}. Similarly, we conclude that there exists a regular isomorphism H' of $A_{(m)}^{*'}(X_{(m)}')$ onto $A_{(2m)}^*(X_{(2m)})$, defined over K_{2m}, so that $H'^{-1}H$ is a regular isomorphism of $A_{(m)}^*(X_{(m)})$ onto $A_{(m)}^{*'}(X_{(m)}')$, defined over K_{2m}. According to AF, Theorem 3, Corollary 1', there exists an Abelian variety A_0, defined over K, and a regular isomorphism H_0 of $A_{(m)}^*(X_{(m)})$ onto A_0, defined over K_m; it follows that H_0GR_m is a rational homomorphism of A_m^* onto A_0, defined over K_m, and, according to AF, Theorem 2, Corollary 1, H_0GR_m is also defined over $K(u_m)$. Since A is the K-image of A_m^* over $K(u_m)$, there exists a rational homomorphism H_1 of A into A_0, defined over K, such that $H_0GR_m = H_1F_m$ or $GR_m = H_0^{-1}H_1F_m$; we have, then, the relations $T_{(m)}H_0^{-1}H_1F_m = T_{(m)}GR_m = G_1T_mR_m = G_1F_m$ and hence $T_{(m)}H_0^{-1}H_1 = G_1$, which shows that $T_{(m)}$ is a regular isomorphism. This concludes the proof of our theorem.

COROLLARY. *Let $K(u)$ be a regular extension of K, and let A^* be an Abelian variety defined over $K(u)$; let A be the K-image of A^* over $K(u)$, and let F be the canonical homomorphism of A^* onto A. If K_1 is any extension of K, independent with respect to $K(u)$ over K, then A is also the K_1-image of A^* over $K_1(u)$, and F is also the corresponding canonical homomorphism.*

Proof: If $\bar{A}$ is the K_1-image of A^* over $K_1(u)$ and if we denote by $\bar{F}$ the canonical homomorphism of A^* onto $\bar{A}$, then, according to AF, Theorem 4, Corollary, there exists a rational isomorphism I of $\bar{A}$ onto A such that $F = I\bar{F}$; if we denote by $\bar{F}_{(m)}$ the rational homomorphism of $A_{(m)}^*$ onto $\bar{A}$ defined by the correspondence

$(x_1, \ldots, x_m) \to \sum_{i=1}^{m} \bar{F}_i(x_i)$, where $(x_t, \ldots, x_m)$ is now a generic point of $A^*_{(m)}$ over $K_1(u_1, \ldots, u_m)$, then we have the relation $F_{(m)} = I\bar{F}_{(m)}$. Since the homomorphism $F_{(m)}$ is regular for sufficiently large m, we conclude that the isomorphism I must be regular.

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[1] "Abelian Varieties over Function Fields," *Trans. Am. Math. Soc.*, **78**, 253, 1955. We shall assume that the reader is familiar with the contents of this paper, which we shall cite as "AF."

[2] "On the Quotient Variety of an Abelian Variety," these PROCEEDINGS, **38**, 1040, 1952, Theorem 1. We observe that Theorem 1 (as well as Theorem 2) in this paper and its proof hold without any modifications also for the more general case where (in the notation used there) the kernel of H contains X.

THE STRONG LAW OF LARGE NUMBERS WHEN THE FIRST MOMENT DOES NOT EXIST

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Let X be a random variable with distribution function F. It is customary to define $EX = +\infty$ if and only if

$$\int_{-\infty}^{0} |x| \, dF(x) < \infty, \qquad \int_{0}^{\infty} x \, dF(x) = \infty. \tag{1}$$

It follows by truncation from the strong law of large numbers for finite EX that if $\{X_n\}$ is a sequence of independent random variables with common distribution function F for which relation (1) holds and if $S_n = \sum_{1}^{n} X_i$, then, as $n \to \infty$,

$$\Pr\left[\frac{S_n}{n} \to +\infty\right] = 1. \tag{2}$$

However, equation (2) can hold even though

$$\int_{-\infty}^{0} |x| \, dF(x) = \infty, \qquad \int_{0}^{\infty} x \, dF(x) = \infty, \tag{3}$$

as is shown by the following:

THEOREM. *If for some constants* $0 < \alpha < \beta < 1$ *and* $C > 0$,

$$F(x) \leq 1 - \frac{C}{x^{\alpha}} \text{ for large positive } x \tag{4}$$

(which implies that

$$\int_{0}^{\infty} x^{\alpha} \, dF(x) = \infty), \tag{4'}$$

while

$$\int_{-\infty}^{0} |x|^{\beta} \, dF(x) < \infty, \tag{5}$$

then equation (2) *holds.*

Proof: We may assume that $0 < \Pr[X > 0] < 1$ and hence that, with probability 1, there will be infinitely many positive and infinitely many negative terms in the sequence $\{X_n\}$. Let Y_n and Z_n denote, respectively, the nth positive and the nth negative term in the sequence $\{X_n\}$. The random variables Y_n and Z_n are independent, with distribution functions which we shall denote, respectively, by G and H, and equation (4) holds with F replaced by G and relation (5) with F replaced by H. The latter fact implies, by a theorem of Marcinkiewicz, that

$$\Pr\left[\sum_1^n \frac{Z_i}{n^{1/\beta}} \to 0\right] = 1, \tag{6}$$

while the former implies that

$$\Pr\left[\sum_1^n Y_i \leq n^{1/\beta}\right] \leq \Pr[\max(Y_1, \ldots, Y_n) \leq n^{1/\beta}] = [G(n^{1/\beta})]^n$$

$$\leq \left(1 - \frac{C}{n^{\alpha/\beta}}\right)^n \leq e^{-Cn^\gamma}; \qquad \gamma = \frac{(\beta - \alpha)}{\beta} > 0. \tag{7}$$

Since $\sum_1^\infty e^{-Cn^\gamma} < \infty$, the Borel-Cantelli lemma shows that

$$\Pr\left[\sum_1^n Y_i > n^{1/\beta} \text{ for sufficiently large } n\right] = 1. \tag{8}$$

From equations (6) and (8) it follows easily that, with probability 1,

$$\frac{S_n}{n} = \sum_1^n \frac{Y_i}{n} + \sum_1^n \frac{Z_i}{n} \to +\infty, \tag{9}$$

which proves the theorem. We remark that relation (4) was used only to obtain the condition

$$\sum_1^n [G(n^{1/\beta})]^n < \infty. \tag{10}$$

It would be desirable to find simple necessary and conditions for equation (2) in terms of F. We do not know whether relations (4′) and (5) are sufficient for equation (2).

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CESÀRO SUMMABILITY OF WALSH-FOURIER SERIES

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The Fejér-Lebesgue theorem asserts that the trigonometric Fourier series of an integrable function is $(C, 1)$ summable almost everywhere, and it is known that the same result holds for (C, α) summability, $\alpha > 0$. In this note I prove the analogous theorem for Walsh-Fourier series. For $f \in L^p$, $p > 1$, a proof was indicated by Paley.[1] In the trigonometric case the estimate $K_n(t) = 0(1/nt^2)$ for the Fejér kernel (and a similar one for $K_n^{(\alpha)}(t)$) makes the proof relatively easy, but as pointed out in an earlier paper,[2] this estimate fails for the Walsh system. It can be shown that the Abel kernel also behaves badly in this respect. Nevertheless, the difficulty can be overcome by a close analysis of the kernel and by the crucial Lemma 3, which may be of some independent interest. For notation and background material, the reader is referred to the earlier paper.[2]

LEMMA 1. *Let $D_n(t)$, $K_n(t)$, and $K_n^{(\alpha)}(t)$ denote the Dirichlet, Fejér, and (C, α) kernels, respectively, for Walsh-Fourier series. If, for a fixed x and $f \in L$,*

$$\int_0^1 D_{2^n}(t)\,|f(x \dotplus t) - f(x)|\,dt \to 0, \tag{1}$$

$$\int_0^1 K_{2^n}(t)\,|f(x \dotplus t) - f(x)|\,dt \to 0, \tag{2}$$

then, for every $\alpha > 0$,

$$\int_0^1 |K_n^{(\alpha)}(t)|\;|f(x \dotplus t) - f(x)|\,dt \to 0,$$

and $\sigma_n^{(\alpha)}(x; f) \to f(x)$.

Proof: For $\alpha = 1$, this is essentially proved in Theorem XVII of the earlier paper.[2] We may therefore assume that $0 < \alpha < 1$, and then the proof follows from the case $\alpha = 1$ exactly as in Theorem 4 of a paper of Yano.[3]

LEMMA 2. *For $n \geq 0$,*

$$K_{2^n}(t) = \left(\frac{1}{2} + \frac{1}{2^{n+1}}\right) D_{2^n}(t) + \frac{1}{4}\sum_{j=1}^{n} 2^{j-n} D_{2^n}(t \dotplus 2^{-j}). \tag{3}$$

Proof: In the proof of Lemma 3 of the earlier paper[2] we have the formula

$$2^{n+1}K_{2^{n+1}}(t) = (1 + \psi_{2^n}(t))2^n K_{2^n}(t) + 2^n D_{2^n}(t).$$

If, in addition, we use the easily proved formulas

$$\begin{aligned} D_{2^{n+1}}(t) &= (1 + \psi_{2^n}(t))D_{2^n}(t), \\ D_{2^{n+1}}(t \dotplus 2^{-j}) &= (1 + \psi_{2^n}(t))D_{2^n}(t \dotplus 2^{-j}) \qquad (1 \leq j \leq n), \\ D_{2^{n+1}}(t \dotplus 2^{-n-1}) &= (1 - \psi_{2^n}(t))D_{2^n}(t), \end{aligned}$$

then equation (3) follows readily by induction. Lemma 2 is equivalent to Lemma 1 of another paper of Yano,[4] where the proof is somewhat more complicated than the one sketched here.

LEMMA 3. *Let C be a measurable subset of $[0, 1]$, $D(x) = \rho(x, C)$, the distance from x to C, and let $h_1 \geq h_2 \geq h_3 \geq \ldots \geq 0$ be a sequence satisfying*

$$\sum_{h_j \leq \delta} h_j \leq M\delta, \tag{4}$$

$$\sum_{h_j > \delta} \frac{1}{h_j} \leq \frac{M}{\delta}. \tag{5}$$

for a constant M and all $\delta > 0$. Then, for almost all $x \in C$, and for any choices of $\pm$,

$$\varphi(x) = \sum_{j=1}^{\infty} \frac{D(x \pm h_j)}{h_j} < \infty. \tag{6}$$

Proof: Without loss of generality, we may take C to be closed. Let $V = (-h_1, 1 + h_1) - C = \sum_{n \geq 1} I_n$, where the I_n are disjoint open intervals of lengths δ_n. For any h such that $0 < |h| \leq h_1$,

$$\int_C D(x + h)\, dx = \int_{C+h} D(x)\, dx = \int_{(C+h)V} D(x)\, dx$$

$$= \sum_{n \geq 1} \int_{(C+h)I_n} D(x)\, dx = \sum_{\delta_n < |h|} + \sum_{\delta_n \geq |h|} = T_1(h) + T_2(h),$$

say. For $\delta_n < |h|$, $|(C + h)I_n| \leq \delta_n$, and on this set $D(x) \leq \delta_n$; so that

$$T_1(h) \leq \sum_{\delta_n < |h|} {\delta_n}^2.$$

Hence

$$\sum_{j=1}^{\infty} \frac{1}{h_j} T_1(\pm h_j) \leq \sum_{j=1}^{\infty} \frac{1}{h_j} \sum_{\delta_n < h_j} {\delta_n}^2 = \sum_{n=1}^{\infty} {\delta_n}^2 \sum_{h_j > \delta_n}^{\infty} \frac{1}{h_j} \leq M \sum_{n=1}^{\infty} \delta_n < \infty.$$

For $\delta_n \geq |h|$, $|(C + h)I_n| \leq |h|$, and on this set $D(x) \leq |h|$, so that

$$T_2(h) \leq h^2 \sum_{\delta_n \geq |h|} 1.$$

Hence

$$\sum_{j=1}^{\infty} \frac{1}{h_j} T_2(\pm h_j) \leq \sum_{j=1}^{\infty} h_j \sum_{\delta_n \geq h_j} 1 = \sum_{n=1}^{\infty} \sum_{h_j \geq \delta_n} h_j \leq M \sum_{n=1}^{\infty} \delta_n < \infty.$$

Therefore

$$\int_C \sum_{j=1}^{\infty} \frac{D(x \pm h_j)}{h_j}\, dx = \sum_{j=1}^{\infty} \frac{1}{h_j} (T_1(\pm h_j) + T_2(\pm h_j)) < \infty;$$

hence the integrand $\varphi(x)$ converges a.e. on C, and the lemma is proved.

We remark that each of formulas (4) and (5) is equivalent to the existence of $r \geq 1$ and q, $0 < q < 1$, such that $h_{j+r} \leq q h_j$ $(j \geq 1)$. In particular, the lemma applies to $h_j = 2^{-j}$.

We are now ready to state and prove our theorem.

THEOREM. *For $\alpha > 0$, the Walsh-Fourier series of an integrable function $f(x)$ is (C, α) summable to $f(x)$ almost everywhere.*

Proof: Let $\alpha_n(x) = r\cdot 2^{-n} \leq x < (r+1)2^{-n} = \beta_n(x)$. Then

$$\int_0^1 D_{2^n}(t)\,|f(x \dotplus t) - f(x)|\,dt = 2^n \int_{\alpha_n(x)}^{\beta_n(x)} |f(t) - f(x)|\,dt = o(1)$$

almost everywhere, by Lebesgue's theorem. By Lemma 1, it is sufficient to show that formula (2) holds a.e. Fix $\eta > 0$. By Lusin's theorem, there is a set C_1 of measure greater than $1 - \eta/2$ on which f is continuous relative to C_1. Again, by Lebesgue's theorem,

$$\lim_{h\to 0} \frac{1}{h} \int_x^{x+h} |f(t) - f(x)|\,dt = 0 \qquad \text{(a.e.).} \quad (7)$$

Applying Egoroff's theorem, we find a set C_2 of measure greater than $1 - \eta/2$ on which equation (7) holds uniformly. Let C be a closed subset of $C_1 \cap C_2$ of measure greater than $1 - \eta$. Thus, on C, we have f uniformly continuous and bounded, say $|f(x)| \leq B$, and if

$$\omega(h) = \sup_{|u| \leq h} \sup_{y \in C} \frac{1}{u} \int_y^{y+u} |f(t) - f(y)|\,dt, \quad (8)$$

we have $\omega(h) \to 0$ as $h \to 0$.

For $x \in C$, multiply equation (3) by $|f(x \dotplus t) - f(x)|$ and integrate, to get

$$\int_0^1 K_{2^n}(t)\,|f(x \dotplus t) - f(x)|\,dt$$

$$= o(1) + \frac{1}{4} \sum_{j=1}^{n} 2^{j-n} \int_0^1 D_{2^n}(t \dotplus 2^{-j})\,|f(x \dotplus t) - f(x)|\,dt$$

$$= o(1) + \frac{1}{4} \sum_{j=1}^{n} 2^{j-n} \int_0^1 D_{2^n}(t)\,|f(x \dotplus 2^{-j} \dotplus t) - f(x)|\,dt.$$

Thus, to prove that formula (2) holds a.e., it is sufficient to show that

$$\Delta_n(x) = \sum_{j=1}^{n} 2^j \int_{\alpha_n(x+2^{-j})}^{\beta_n(x+2^{-j})} |f(t) - f(x)|\,dt \quad (9)$$

tends to zero almost everywhere on C, since η is arbitrary.

Fix $\epsilon > 0$, then choose $p = p(\epsilon)$ so that $\omega(2^{-p}) < \epsilon$ and so that $|f(y) - f(x)| < \epsilon$ for all $x, y \in C$ with $|x - y| \leq 2^{-p}$. Write

$$\Delta_n(x) = \sum_{j=1}^{p} + \sum_{j=p+1}^{n} = S_1 + S_2, \quad (10)$$

say. We have

$$S_1 \leq 2^{p+1} \left[\max_{0 \leq r < 2^n} \int_{r\cdot 2^{-n}}^{(r+1)2^{-n}} |f(t)|\,dt + B\cdot 2^{-n} \right] < \epsilon \quad (11)$$

for $n > n_0$, $x \in C$.

For $p < j \leq n$ and $x \in C$, define $y_j = y_j(x)$ to be a point of C closest to $z_j = x \dotplus 2^{-j}$. Then

$$\int_{\alpha_n(z_j)}^{\beta_n(z_j)} |f(t) - f(x)|\,dt \leq \int_{\alpha_n(z_j)}^{\beta_n(z_j)} |f(t) - f(y_j)|\,dt + \int_{\alpha_n(z_j)}^{\beta_n(z_j)} |f(y_j) - f(x)|\,dt. \quad (12)$$

Since $x, y_j \in C$ and $|x - y_j| \leq 2|x - z_j| = 2^{1-j} \leq 2^{-p}$, the second integral on the right of formula (12) is less than $2^{-n}\epsilon$. The first integral is equal to

$$\int_{\alpha_n(z_j)}^{y_j} + -\int_{y_j}^{\beta_n(z_j)} |f(t) - f(y_j)| \leq |y_j - \alpha_n(z_j)|\,\omega(|y_j - \alpha_n(z_j)|) + |\beta_n(z_j) - y_j|\,\omega(|\beta_n(z_j) - y_j|) \leq 2v_j\omega(v_j),$$

where $v_j = \max(|y_j - \alpha_n(z_j)|, |\beta_n(z_j) - y_j|)$. Now $v_j \leq \rho(z_j, C) + 2^{-n} \leq 2^{1-j} \leq 2^{-p}$, so $\omega(v_j) < \epsilon$. Hence the first integral is dominated by $2\epsilon(\rho(z_j, C) + 2^{-n})$, and

$$\int_{\alpha_n(z_j)}^{\beta_n(z_j)} |f(t) - f(x)|\, dt < 3\epsilon(\rho(z_j, C) + 2^{-n}). \tag{13}$$

Multiplying formula (13) by 2^j and summing, we find

$$S_2 < 6\epsilon + 3\epsilon \sum_{j=p+1}^{n} 2^j\rho(z_j, C),$$

$$\Delta_n(x) < 7\epsilon + 3\epsilon \sum_{j=1}^{\infty} 2^j\rho(x \dot{+} 2^{-j}, C),$$

for $n > n_0$ and $x \in C$. Applying Lemma 3, with $h_j = 2^{-j}$, we conclude that $\Delta_n(x) \to 0$ a.e. on C, and the theorem is proved.

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[2] N. J. Fine, "On the Walsh Functions," *Trans. Am. Math. Soc.*, **65**, No. 3, 372–414, 1949.

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*GENERALIZED DERIVATIVES AND EXPANSIONS**

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I. *Derivatives and Expansions of a Function with Regard to a Family of Systems of Numbers.*—Consider a system B of n (real or complex) numbers, not necessarily distinct. For any function, f, having the necessary properties of definition and differentiability at the points of B, let $f[B]$ denote the interpolation polynomial of f at B, i.e., the polynomial of degree $\leqslant n - 1$ such that $f[B]$ and f have equal values $f[B](b) = f(b)$, at each point b in B and that, if b is of multiplicity $m > 1$ in B, also the derivatives of $f[B]$ and f up to the $(m - 1)$th have equal values[1] at b.

Let B denote a one-parameter family of n-tuples of not necessarily distinct numbers, and $B(t)$ the member of that family corresponding to the (real or complex) number t. Let f be a function such that $f[B(t)]$ is defined for every t in a neighborhood of t_0. Then we define *the derivative at* t_0 *of* f *with respect to the family* B as the polynomial

$$D_{B,t_0}f = \lim_{t \to t_0} \frac{f[B(t)] - f[B(t_0)]}{t - t_0} \quad \text{or} \quad \frac{\partial}{\partial t}(f[B(t)](z))|_{t=t_0}. \tag{1}$$

If, for any t, $B(t)$ consists of the single number t, then B may be regarded as the identity function j assuming the value $j(\mathbf{z}) = \mathbf{z}$ for any $\mathbf{z}$, and each $f[B(t)]$ is the constant polynomial of value $f(t)$. Hence $D_{j,t_0}f$ is the constant polynomial of value $Df(t_0)$, that is, the value at t_0 of the classical derivative of f. More generally, if $n = 1$ and, for some function h having a derivative at t_0, each system $B(t)$ consists of the single number $h(t)$, then $D_{h,t_0}f$ is the constant polynomial whose value is the classical derivative at t_0 of the function of a function, fh.

If $n = 2$ and $B(t) = \{h_1(t), h_2(t)\}$, then, for any t_0 such that $h_1(t_0) \neq h_2(t_0)$, $D_{B,t_0}f$ is the linear polynomial assuming for $\mathbf{z}$ the value

$$D_{B,t_0}f(\mathbf{z}) = D\frac{fh_1 \cdot h_2 - fh_2 \cdot h_1}{h_2 - h_1}(t_0) + D\frac{fh_2 - fh_1}{h_2 - h_1}(t_0) \cdot \mathbf{z}. \tag{2}$$

Here $fh_1 \cdot h_2$ is the product of the function obtained by substituting h_1 into f, multiplied by h_2. The two coefficients of the linear polynomial are the values at t_0 of the classical derivatives of the respective quotients. (These quotients are symmetric in h_1 and h_2. If $h_1 + h_2$ and $h_1 \cdot h_2$ are differentiable at t_0, then one can show that $h_1(t) \neq h_2(t)$ for all t near t_0.)

Now let n be any integer, and B a family of n-tuples. Clearly, for any t_0, $D_{B,t_0}j^k$ is the constant polynomial O, where $j^k(\mathbf{z}) = \mathbf{z}^k$ for any $\mathbf{z}$ and $k = 0, 1, \ldots, n-1$. The interpolation polynomial of j^n at $B(t)$, if one sets

$$p_{B(t)} = \prod_{b \in B(t)} (j - b),$$

is $j^n[B(t)] = j^n - p_{B(t)} = s_1(t) \cdot j^{n-1} + \ldots + (-1)^{n-1}s_n(t)$, where $s_1(t), \ldots, s_n(t)$ are the elementary symmetric functions of the numbers in $B(t)$. Hence $D_{B,t_0}j^n$ exists if and only if the family B has the property that the functions $s_1, \ldots, s_n$ have classical derivatives at t_0. Such a family of n-tuples will be called *derivable* at t_0.

The m-th order derivative at t_0 of f with respect to B can be defined inductively:

$$D^m_{B,t_0}f = \lim_{t \to t_0} \frac{D^{m-1}f[B(t)] - D^{m-1}f[B(t_0)]}{t - t_0}.$$

If the family B of n-tuples is derivable of m-th order at t_0 (that is, if $D^m s_1(t_0), \ldots, D^m s_n(t_0)$ exist), then either of the following conditions is sufficient for the existence of $D^m_{B,t_0}f$:

a) f is regular at (i.e., differentiable in a complex neighborhood of) each number belonging to $B(t_0)$.

b) The values of t as well as the numbers in all systems $B(t)$ are real, and f is a real function such that $D^{(m+1)q-1}f(b)$ exists for any point b in $B(t_0)$ if q is the multiplicity of b in $B(t_0)$.

If $D_{B,t_0}f$ exists, then

$$D_{B,t_0}f(\mathbf{z}) = -\frac{1}{2\pi i}\int_\Gamma \frac{fB^2(t_0)(\mathbf{z}')}{\mathbf{z}' - \mathbf{z}} \cdot \frac{\partial}{\partial t}\left(\frac{p_{B(t)}(\mathbf{z})}{p_{B(t)}(\mathbf{z}')}\right)\Big|_{t=t_0} d\mathbf{z}', \tag{3}$$

where $B^2(t_0)$ is the system in which each number has twice the multiplicity it has in $B(t_0)$ and where, if $B(t_0)$ contains k distinct numbers, Γ consists of k simple closed curves with mutually disjoint interiors each of which encircles one of the

points in $B(t_0)$ once in the counterclockwise sense. If, for any number c, a 2-place function Q is defined by setting

$$Q(t, z') = \frac{p_{B(t)}(c)}{p_{B(t)}(z')},$$

and if $D_1Q(b, j)$ denotes the function obtained by substituting (b, j) into D_1Q (the first-place partial derivative of Q), then equation (3) can be expressed in the form

$$D_{B,a}f(c) = -\frac{1}{2\pi i}\int_\Gamma \left[\frac{f[B^2(a)]}{j - c}\cdot D_1Q(a, j)\right], \tag{3'}$$

where a and c are the constant functions of values a and c, respectively.

The integrand in equation (3) or (3') is a rational function. If $n = 2$ and $B(t) = \{h_1(t), h_2(t)\}$, the linear polynomial (2) can be obtained from equations (3) and (3') without the assumption of differentiability or even continuity of h_1 and h_2 at t_0. Only $h_1 + h_2$ and $h_1 \cdot h_2$ must be differentiable at t_0 to make the family B derivable at t_0.

One easily obtains the following generalized Taylor expansions expressing the interpolation polynomial of f at $B(t)$ as linear combinations of the derivatives of various orders at t_0 of f with respect to the family B, the coefficients being the powers of $t - t_0$ divided by the proper factorials. If f satisfies condition a, then

$$f[B(t)] = \sum_{k=0}^{k=\infty} \frac{1}{k!} D^k_{B,t_0}f\cdot(t - t_0)^k; \tag{4}$$

that is to say,

$$f[B(t)](z) = \sum_{k=0}^{k=\infty} \frac{1}{k!} D^k_{B,t_0}f(z)\cdot(t - t_0)^k, \qquad \text{for any } z. \tag{4'}$$

If all t and all systems $B(t)$ are real and $D^{m+1}_{B,t_1}f$ exists for each t_1 between and including t_0 and t, then

$$f[B(t)] = \sum_{k=0}^{k=m} \frac{1}{k!} D^k_{B,t_0}f\cdot(t - t_0)^k + R_{m+1}, \tag{5}$$

where

$$R_{m+1} = \frac{1}{rm!}(1 - s)^{1+m-r}\cdot D^{m+1}_{B,t^*}f\cdot(t - t_0)^{m+1}$$

for some s between 0 and 1, for $t^* = t_0 + s(t - t_0)$, and for any positive number r. If $B(t)$ consists of the single number t, then equation (5) is essentially[2] the Taylor expansion of f about t_0.

In equation (4) the family B is assumed to be regular at t_0 (that is, the elementary symmetric functions s_j are regular at t_0).

If f is allowed to have isolated singularities at points belonging to $B(0)$, then $f[B(t)]$ is defined for t near 0, and

$$f[B(t)] = \sum_{k=-\infty}^{k=\infty} \frac{1}{2\pi i}\int_\Gamma \frac{f[B(t')]}{(t' - t_0)^{k+1}}\,dt'\cdot(t - t_0)^k. \tag{6}$$

II. *Derivatives and Expansions of a Function with Respect to a Function at a System of Numbers.*—We will call a function g and a finite system A of (not necessarily distinct) numbers *compatible* if each point, a, in A has a neighborhood U_a in which g is analytic without being identical with $g[A]$. Here g may be a composite of unrelated functions in the various neighborhoods U_a. But if g is a polynomial of degree n, then only a system containing at most n numbers is compatible with g.

Consider a compatible pair, g and A. For any given number t, denote by $B(t)$ the system of all those roots z (each counted with its multiplicity) of the equation

$$g(z) - g[A](z) = t \tag{7}$$

that lie in $\sum_{a \in A} U_a$. Clearly, $B(0)$ contains A as a subsystem—possibly as a proper subsystem, since a number may have a higher multiplicity in $B(0)$ than in A. For any number t, $g[B(t)] - g[A]$ is the constant polynomial t of value t, and $g[A] = g[B(0)]$.

Since g and A are compatible, one can determine two positive numbers, d and e, such that, for $|t| \leq e$, all systems $B(t)$ contain the same number of points in the intersection of U_a and the circle U(a, d), for each element, a, of A. The family of all $B(t)$ for $|t| \leq e$ will be denoted by $B\{g, A\}$. This family is regular at $t = 0$.

If $D_{B\{g, A\}, 0}f$ exists, we call this polynomial *the derivative of f with respect to g at the system* A and denote it by $D_g f(A)$. This concept is a twofold generalization of the classical derivative of f at x, which Cauchy denoted by $D_x f(x)$. The x in the subscript, which stands for the identity function j, is replaced by any function, g; the argument x, which stands for a single number, is replaced by any finite system of numbers, A, provided that g and A are compatible.

One can prove that

$$D_g f(A) = \lim_{t \to 0} \frac{f[B(t)] - f[B(0)]}{g[B(t)] - g[B(0)]} = \lim_{t \to 0} \frac{1}{t} (f[B(t)] - f[B(0)]) = D_g f(B(0))$$

and, more generally,

$$D_g^k f(A) = D_{B\{g, A\}, 0}^k f = \lim_{t \to 0} \frac{1}{t} (D_g^{k-1} f[B(t)] - D_g^{k-1} f[B(0)]).$$

The last theorem is a consequence of the following lemma:

$$B\{g, B(t_1)\}(t) = B\{g, B(0)\}(t + t_1) \qquad \text{for} \qquad |t| + |t_1| \leq e.$$

Our theory includes as special cases the expansions of Taylor, Lagrange, Jacobi, Bürmann, Laurent, Teixeira, and Kienast as well as generalizations of these expansions.

For instance, let A be any system of not necessarily distinct numbers and $g = p_A = \prod_{a \in A} (j - a)$. Then, clearly, $g[A] = p_A[A] = O$, and equation (7) reduces to $g(z) = t$. Now let z be any given number that is sufficiently close to some number, a, of A. More precisely, let z be any number in U_a at a distance $\leq d$ from a and such that $|p_A(z)| \leq e$. Then, for $t = p_A(z)$, the system $B(t)$ belongs to the derivable family $B\{p_A, A\}$ and obviously includes the number z. It follows that, for z, any function f assumes the same value as does the interpolation polynomial $f[B(t)]$. Hence, if in equation (4) one chooses $t = p_A(z)$ and $t_0 = 0$, the left side equals $f(z)$,

while the right side is the Jacobi expansion[3] in powers of $p_A(z)$, the coefficients being the values at z of the derivatives of f with respect to p_A at A, divided by the proper factorials; that is to say, under assumption a,

$$f(z) = \sum_{k=0}^{k=\infty} \frac{1}{k!} (D^k_{p_A} f(A))(z) \cdot p^k_A(z) \qquad \text{or} \qquad f = \sum_{k=0}^{k=\infty} \frac{1}{k!} D^k_{p_A} f(A) \cdot p^k_A.$$

Of course, one may also start with any monic polynomial, p, and denote by A_p the system of all zeros of p, in which case

$$f = \sum_{k=0}^{k=\infty} \frac{1}{k!} D^k_p f(A_p) \cdot p^k.$$

Assume that (1) the numbers in A are real or occur in conjugate pairs; (2) f has conjugate values at conjugate arguments; (3) f is $(m + 1)$-fold really differentiable at all single real, and regular at all other, elements of $B\{p_A, A\}(t_1)$ for any t_1 between and including t_0 and t. Then, for any m, equation (5) yields a Jacobi expansion with an $(m + 1)$th remainder. The latter does not seem to have been determined heretofore.

A generalization of the Jacobi expansion results if, for any compatible pair g, A, one sets $t = g(z) - g[A(z)]$ and $t_0 = 0$ in equation (4′). One thus obtains the following expansion in powers of $g - g[A]$:

$$f = \sum_{k=0}^{k=\infty} \frac{1}{k!} D^k_g f(A) \cdot (g - g[A])^k.$$

By setting $g = p_A$, $t = p_A(z)$, and $t_0 = 0$ in equation (6), one obtains the Kienast expansion.[4] If A consists of a single number, then, in the neighborhood of any simple root of equation (7), equation (6) yields the Teixeira expansion.[5]

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[1] We use roman type for numbers (0, 1, i, . . .) and italics for functions (e.g., the constant function O and the identity function j assuming the values $O(z) = 0$ and $j(z) = z$ for any z). Correspondingly, we use roman letters (a, c, t, z, . . .) for numerical variables and italics (f, g, h, . . .) for function variables, that is, symbols that may be replaced by the designations of specific numbers and functions, respectively. In particular, a, b, c, are replaceable by any constant function. (Cf. Menger, *Calculus: A Modern Approach* [Boston, 1955].) Systems of numbers will be designated by capitals (A, B, . . .) in roman type, families of such systems in italic capitals.

[2] Except that Taylor expresses the number $f(t)$ in terms of the numbers $D^k f(t_0)$ and the powers of $t - t_0$, whereas we express the constant function of value $f(t)$ in terms of the constant functions of values $D^k f(t_0)$ and the powers of $t - t_0$.

[3] C. G. J. Jacobi, *Crelle's J.*, **53**, 103–126, 1856.

[4] A. Kienast, inaugural dissertation, University of Zurich, 1906.

[5] M. F. G. Teixeira, *J. f. Math.*, **122**, 97–123, 1900. Cf. also Whittaker and Watson's presentation in *A Course of Modern Analysis* (4th ed.; Cambridge: At the University Press, 1952), p. 131.

ON THE MODULAR REPRESENTATIONS OF THE SYMMETRIC GROUP VI

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1. In the development of the modular representation theory of S_n many concepts of the general theory have been made explicit. For example, the *p-block* to which a diagram or representation belongs is determined by its p-core, while the *power of* p dividing the degree of $[\lambda]$ is expressible in terms of the p-quotient (star diagram)[1] of $[\lambda]$. In the general theory the one-to-one correspondence which can be established between the indecomposables of the regular representation and the modular irreducible representations is fundamental.[2, 3] To establish this correspondence for S_n, we shall define an *operator* Y_p which, when applied to specified diagrams in a block B, will yield the indecomposables of that block. Thus we have a method for constructing the decomposition matrix[3] D of S_n.

In order to illustrate the ideas which we shall have to describe, we give the D-matrix for the block B for S_8 with 3-core [2], as it appears in the thesis of J. H. Chung.[4]

	1	27	35	7	13	Degree
[8]	1					1
[5, 3]	1	1				28
[5, 2, 1]	2	1	1			64
$[5, 1^3]$			1			35
[4, 3, 1]	1	1	1	1		70
$[3^2, 1^2]$	1		1	1	1	56
$[2^4]$	1				1	14
$[2^2, 1^4]$				1	1	20
$[2, 1^6]$				1		7

A *p-regular* diagram[5] is defined to be one having no p rows of equal length. Thus [8], [5, 3], [5, 2, 1], [4, 3, 1], and $[3^2, 1^2]$ are 3-regular, and we call them *heads* in B. But, equally, [7, 1], [6, 2], $[4^2]$, [4, 2^2], [3, 2^2, 1] are heads in the block B' with core $[1^2]$. We call the associated diagrams $[2, 1^6]$, $[2^2, 1^4]$, $[2^4]$, $[3^2, 1^2]$, [4, 3, 1], *feet* in B. The operator Y_p establishes a one-to-one correspondence between the head and foot of an indecomposable in B, and, if the diagrams are arranged in dictionary order, all those belonging to a given indecomposable will lie *between* such a head and foot.

2. Consider now the significance of p relative to the representation theory of S_n. If $p > n$, every partition of n yields a Young diagram which is a p-core and so an indecomposable of the regular representation. On the other hand, if $p = 1$, all the representations of S_n belong to one block, and there is a single "indecomposable," namely, the regular representation of S_n. In this case we *do* have an operator which yields the irreducible components with the required multiplicity.[6]

In fact, the *raising operator* Y of Young, when applied to $[1^n]$, yields precisely the f_λ standard diagrams $[\lambda]$, each of which represents one irreducible component $[\lambda]$ of the regular representation.

For $p > 1$ let us consider this raising operator applied to a foot diagram F in the block B. From the fact that all the diagrams of B have the same *p-content*[7] (i.e., the same number of residues of each class appearing in their p-graphs[8, 9]), it is natural to require (i) *that the raising operator leave the residue class of each symbol invariant.* This restriction alone is sufficient to generate many indecomposables from their foot diagrams, but trouble arises, e.g., for $p = 3$ in the case of $F = [4, 3, 1]$, where $[5, 3]$ can be obtained in two different ways. To overcome this difficulty, we write F in the form

$$\begin{matrix} a_0\ a_1\ a_2\ a_0 \\ b_2\ b_0\ b_1 \\ c_1 \end{matrix}$$

where the residue class of any position is affixed as a subscript. The redundancy in the operator arises when there is a vacant r-position[9] *above* at least two removable r-nodes of F; in such circumstances we require (ii) *that the removable r-nodes shall all be assigned the symbol of the lowest one in F.* In our example this sets $b_1 = c_1$, and the redundancy disappears:

$$\begin{matrix} a_0\ a_1\ a_2\ a_0 \\ b_2\ b_0\ c_1 \\ c_1 \end{matrix} + \begin{matrix} a_0\ a_1\ a_2\ a_0\ c_1 \\ b_2\ b_0 \\ c_1 \end{matrix} + \begin{matrix} a_0\ a_1\ a_2\ a_0\ c_1 \\ b_2\ b_0\ c_1 \end{matrix}$$

The same sort of redundancy arises in studying the r-Boolean Algebra of a core[9], in which case all the elements of a given "dimension" have the same multiplicity and constitute an indecomposable when counted once only. Our example is, in fact, the set of elements of dimension two in the 1-BA arising from $[4,2]$ for $p = 3$.

Since every foot diagram F is obtainable by r-inducing from some foot diagram $\bar{F}$, the identification (ii) in $\bar{F}$ must be continued in F in addition to any new identification which may arise. If we denote the operator Y subject to (i) and (ii) by Y_p, then we have the following:

THEOREM. *By applying Y_p to the foot diagrams in any block, we generate the indecomposables of that block and so the D-matrix.*

The proof of this result involves an induction based on the Nakayama reciprocity formulas[3] and will be given elsewhere.

3. It has already been remarked that all the ordinary representations belonging to an indecomposable "admit" the same permutations of the residues making up the common content, and the number of such permutations is equal to the degree of the corresponding modular representation.[8, 10] But an admitted permutation does not characterize an indecomposable, since it may be admitted by several indecomposables. However, the pattern of admitted permutations does correspond to the multiplicities with which the indecomposables of S_n arise as a result of r-inducing on those of S_{n-1}.

To establish the one-to-one correspondence between the indecomposables and the modular irreducibles envisaged in the general theory, it is sufficient to attach to the last residue in a permutation, i.e., the r of the latest r-inducing, a literal subscript

indicating the position (subject to restriction (ii)) occupied by that residue in the foot diagram F of the indecomposable in question. This identifies uniquely a standard diagram in each component of the indecomposable, assuming that the identification has been similarly made for S_{n-1}. Thus we can "peel off" the modular irreducibles from left to right in the D-matrix.

In brief, this study of the modular representation theory amounts to a detailed analysis of the *standardness condition* in relation to the *raising operator* and the *inducing process*. Anomalies in the operator are associated with anomalies in the standardness condition and vice versa. For example, if R denotes the raising of a symbol from the second row to the first, and we 2-induce with $p = 3$, we have

$$\begin{matrix} 1\,2 \\ 3\,4 \end{matrix} \xrightarrow{R^2} 1\,2\,3\,4, \qquad \begin{matrix} 1\,2\,5 \\ 3\,4 \end{matrix} \xrightarrow{R} \begin{matrix} 1\,2\,3\,4, \\ 5 \end{matrix}$$

indicating that $R = R\,(R \cdot R^{-1})$, where the parenthesis interchanges 3 and 5 on the right. On the left the diagrams are associated with the identity representation and the permutation 0120, while on the right they are associated with the same variable of the modular irreducible representation [4, 1] and the permutation 01202. For $n \leq 2p$ a permutation uniquely determines a standard diagram; hence no literal subscript is necessary.

We remark in conclusion that the relation $C = D'D$ leads to the interpretation of the Cartan invariants[3] of S_n in terms of the operator Y_p.

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THE VALIDITY OF KRON'S METHOD OF TEARING*

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In a preceding paper[1] we defined the network problem in purely algebraic-topological terms and gave a sufficient condition for the existence and uniqueness of a solution. We consider the network as a 1-complex, and the problem of solving the network may be described as an untangling of a twisted isomorphism between the homology and cohomology sequences of K modulo its zero skeleton. This twisted isomorphism is defined as follows: First, there is given a power-definite isomorphism L between the group of 1-chains and the group of 1-cochains of K; this isomorphism is independent of the topological structure of the complex. It is, let us say, given by the physical problem at hand. This isomorphism, however, *induces* a twisted isomorphism (cf. diagram of first paper[1]) of the homology and cohomology sequences which *does* depend upon the topological structure. Kron's method[2] of tearing consists essentially in deducing from the solution of one (easier to solve) network $\tilde{K}$ the solution for a network K having the same number of branches as K and the same isomorphism L between the groups of 1-chains and 1-cochains. We interpret this process in terms of the transformations induced by the simplicial mapping of $\tilde{K}$ on K. In so doing, we obtain a complete proof for the validity of this highly important method.

Let K be a 1-complex and L a power-definite isomorphism of $C^1(K)$ on $C_1(K)$. The complex K and the isomorphism L define the diagram of homomorphisms pictured in the companion paper. Now let $\tilde{K}$ be another complex such that there is a simplicial mapping, φ, of $\tilde{K}$ on K which induces an isomorphism of $C^1(\tilde{K})$ on $C^1(K)$. The system of transformations *induced* by φ on the algebraic structures may be represented by Figure 1. In this diagram, H^1, C^1, P^0 denote $H^1(K)$, $C^1(K)$, $P^0(K)$; and H_1, C_1, P_0 denote $H_1(K)$, $C_1(K)$, $P_0(K)$. Likewise, $\tilde{H}^1$, $\tilde{C}^1$, $\tilde{P}^0$ denote $H^1(\tilde{K})$, $C^1(\tilde{K})$, $P^0(\tilde{K})$; and $\tilde{H}_1$, $\tilde{C}_1$, $\tilde{P}_0$ stand for $H_1(\tilde{K})$, $C_1(\tilde{K})$, $P_0(\tilde{K})$.

The system of mappings contained in the triangle whose base is the left edge and whose opposite vertex is the 0 in the center of the diagram is a reproduction[1] of the algebraic network diagram of K. That is, the sequence of mappings along the diagonal from the upper left-hand corner to the center is the reduced homology sequence of K modulo its zero skeleton K^0, and that along the diagonal from the lower left-hand corner to the center is its reduced cohomology sequence, while the vertical mappings L, L' downward and Y, Y' upward are just the same as in the diagram of the preceding paper.[1]

Likewise, the system of mappings contained in the triangle whose base is the right edge and opposite vertex is the 0 in the center of the diagram is a reproduction of the algebraic network diagram of $\tilde{K}$. Tildes are placed over the corresponding mappings to distinguish these from those of the K system. The horizontal mappings P, Q, R are the homomorphisms induced by the simplicial mapping of $\tilde{K}$ on K, on the corresponding chain groups, while P_t, Q_t, R_t denote the induced homomorphisms of the cochain groups. $\tilde{L}$ is *defined* as Q_tLQ, $\tilde{Y} = \tilde{L}^{-1}$, $\tilde{L}' = \tilde{C}_t\tilde{L}\tilde{C}$, $\tilde{Y}' = \tilde{\delta}\tilde{Y}\tilde{\delta}$. Commutativity holds for all paths not going through 0.

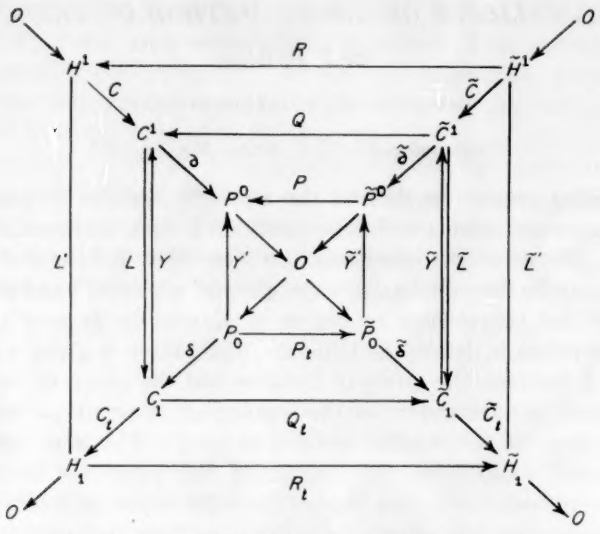

FIG. 1.—The Squirrel-cage diagram.

Now let a network problem be given for K, that is, let $I' \in P^0$, $e' \in H_1$ be given. We will show that these elements uniquely determine a network problem for $\tilde{K}$ and thus that the solution for the network problem of K may be obtained from the solution of the induced network problem $\tilde{K}$ by means of the isomorphisms Q and Q_t. Now I' and e' uniquely determine the required elements J of C^1 and V of C_1. Then we define $\tilde{e}' = R_t e'$, for R_t maps H_1 onto $\tilde{H}_1$, and we define $\tilde{I}'$ as $Q^{-1}J$. It is clear that $\tilde{L}$ is also power definite, and so $\tilde{I}'$ and $\tilde{e}'$ uniquely determine the solutions $\tilde{J}$ and $\tilde{V}$ by the theorem of the first paper. These may be computed directly in terms of $\tilde{I}'$ and $\tilde{e}'$. And then, of course, J and V may be obtained directly from $\tilde{J}$ and $\tilde{V}$. This argument establishes the validity of the method of tearing and interconnecting. Discussion of the explicit construction of the solution via tearing will be given elsewhere.

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NECESSARY AND SUFFICIENT CONDITIONS FOR CARLSON'S THEOREM ON ENTIRE FUNCTIONS

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1. *Introduction.*—It is our main purpose to outline a proof of the following theorem, for which only the necessity of condition (1.4) has previously been proved. It has long been suspected that (1.4) is not sufficient.

THEOREM 1. *In order that each entire function $f(z)$ satisfying the conditions*

$$f(z) = 0(1)e^{\tau|z|} \quad \text{for some } \tau < \infty, \tag{1.1}$$

$$f(iy) = 0(1)e^{c|y|} \quad \text{for some } c < \pi, \tag{1.2}$$

$$f(n) = 0 \quad \text{for each } n \text{ in } A, \tag{1.3}$$

vanish identically, it is necessary and sufficient that

$$\bar{D}(A) = 1, \tag{1.4}$$

where $\bar{D}(A)$ is the upper density of the set A.

The upper density $\bar{D}(A)$ is defined as usual by the formula

$$\bar{D}(A) = \limsup_{t \to \infty} \frac{A(t)}{t}, \tag{1.5}$$

where $A(t)$ is the number of integers n in A such that $0 < n \leq t$. Theorem 1 provides an optimal extension of a fundamental theorem of Carlson which states that if $f(z)$ is an entire function satisfying (1.1) and (1.2) and if $f(n) = 0$ for each positive integer n, then $f(z) \equiv 0$. W. H. J. Fuchs[1] has obtained a necessary and sufficient condition that each function $f(z)$ regular in the right half-plane and satisfying (1.1), (1.2), and (1.3) vanish identically. His condition is different from ours, so that there exists a set A of positive integers satisfying (1.4) but not Fuchs's condition.

Our proof of Theorem 1 will depend upon $\bar{D}_L(A)$, a modified logarithmic density of A, which is defined by

$$\bar{D}_L(A) = \inf_{\lambda > 1} \limsup_{x \to \infty} \frac{1}{\log \lambda} \sum_{n=x}^{\lambda x}{}^{*} \frac{1}{n}. \tag{1.6}$$

In (1.6) and formulas which follow, a star on a $\sum$ indicates that the index of summation is restricted to lie in A. We prove

THEOREM 2. *Theorem* 1 *remains valid if we replace* (1.4) *by the condition* $\bar{D}_L(A) = 1$.

THEOREM 3. *For any set A of positive integers, $\bar{D}(A) = 1$ if and only if $\bar{D}_L(A) = 1$.*

Theorem 1 then follows from Theorems 2 and 3.

2. *Proof of the Sufficiency of the Condition $\bar{D}_L(A) = 1$.*—We now assume that $\bar{D}_L(A) = 1$ and prove that each entire function satisfying (1.1), (1.2), and (1.3) must vanish identically. Suppose, on the contrary, that there exists an entire function $f(z)$ satisfying (1.1), (1.2), and (1.3) for which $f(z) \not\equiv 0$. There is no loss of generality in assuming that $f(0) = 1$. Let the zeros of $f(z)$ be denoted by z_1,

$z_2, \ldots$, where $z_n = r_n \exp(i\theta_n)$. A modification of Carleman's theorem yields the inequality

$$\sum_{n=t}^{\lambda t}{}^{*} \frac{1}{n} \leq (\lambda t)^{-2} \sum_{r_n \leq \lambda t} r_n + \frac{1}{\pi \lambda t} \int_{-\pi/2}^{\pi/2} \log |f(\lambda t e^{i\theta})| \cos\theta \, d\theta - \frac{1}{\pi t} \int_{-\pi/2}^{\pi/2} \log |f(te^{i\theta})| \cos\theta \, d\theta + \frac{1}{2\pi} \int_t^{\lambda t} \{y^{-2} - (\lambda t)^{-2}\} \log |f(iy) f(-iy)| \, dy + \frac{1}{2\pi} \int_0^t \{t^{-2} - (\lambda t)^{-2}\} \log |f(iy) f(-iy)| \, dy = \sum_1 + \int_1 - \int_2 + \int_3 + \int_4. \quad (2.1)$$

Known techniques[2] yield the estimates $\sum_1 \leq K$, $\int_1 \leq K$, $\int_2 \geq -K$, $\int_3 \leq (c/\pi) \log \lambda + K$, $\int_4 \leq K$. Here $K = K(c, \tau, t)$ is independent of λ and is bounded for large t. Applying these estimates to (2.1) and letting first t and then λ approach infinity, we see that $\bar{D}_L(A) \leq (c/\pi) < 1$, contradicting our hypothesis.

3. *Proof of the Necessity of the Condition* $\bar{D}_L(A) = 1$.—We now assume that each entire function $f(z)$ satisfying (1.1), (1.2), and (1.3) vanishes identically, and prove that $\bar{D}_L(A) = 1$. Suppose, on the contrary, that $\bar{D}_L(A) < 1$. We prove the following lemma.

LEMMA 1. *If* $\bar{D}_L(A) < 1$, *then* $\bar{D}(A) < 1$.

To prove this lemma, we choose a number ψ for which $\bar{D}_L(A) < \psi < 1$. We may choose a number $\lambda > 1$ and then a number $M > 0$, so that

$$\sum_{n=t}^{\lambda t}{}^{*} \frac{1}{n} < \psi \log \lambda \qquad \text{for } t > M. \quad (3.1)$$

We shall prove that

$$\bar{D}(A) \leq \frac{\lambda - \lambda^{1-\psi}}{\lambda - 1} < 1. \quad (3.2)$$

Let $t > M$, and put $t = \alpha\lambda^p$, where $M \leq \alpha < \lambda M$ and p is a nonnegative integer. Put $B_k = A(\alpha\lambda^{k+1}) - A(\alpha\lambda^k)$, $k = 0, 1, \ldots, p-1$, so that $A(t) \leq \alpha + \sum_{k=0}^{p-1} B_k$. Using (3.1) to estimate the B_k, we find that

$$\psi \log \lambda \geq \sum_{\alpha\lambda^k}^{\alpha\lambda^{k+1}}{}^{*} \frac{1}{n} \geq \sum_{\alpha\lambda^{k+1} - B_k}^{\alpha\lambda^{k+1}} \frac{1}{n} \geq \log \frac{\alpha\lambda^{k+1}}{\alpha\lambda^{k+1} - B_k + 2},$$

which implies that $B_k \leq 2 + \alpha\lambda^{k+1}\{1 - \lambda^{-\psi}\}$, $A(t) = A(\alpha\lambda^p) \leq \alpha\{1 - \lambda^{-\psi}\}$ $\lambda(\lambda^p - 1)/(\lambda - 1) + \alpha + 2p$, and hence that (3.2) holds. This completes the proof of the lemma.

The conclusion $\bar{D}(A) < 1$ implies[3] that the function $f_A(z)$, defined by

$$f_A(z) = \prod_{n \in A} \left(1 - \frac{z^2}{n^2}\right),$$

satisfies (1.1), (1.2), and (1.3) and therefore violates our hypothesis, establishing the necessity of the condition $\bar{D}_L(A) = 1$.

4. *Proof of Theorems* 1 *and* 3.—We can now complete the proofs of Theorems 1 and 3 by proving Theorem 3. First, suppose that $\bar{D}(A) = 1$. Then Lemma 1 implies that $\bar{D}_L(A) = 1$. Suppose, next, that $\bar{D}_L(A) = 1$. Then each entire func-

tion satisfying (1.1), (1.2), and (1.3) must vanish identically. But if $\bar{D}(A) < 1$, then the function $f_A(z)$ above satisfies (1.1), (1.2), and (1.3), and the condition $f_A(0) = 1$. Hence $\bar{D}(A) = 1$. This completes the proofs of the theorems.

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CONTENTS

Page

BIOCHEMISTRY.—THE EFFECT OF THIOCTIC ACID ON THE QUANTUM EFFICIENCY OF THE HILL REACTION IN INTERMITTENT LIGHT . . . . *By D. F. Bradley and M. Calvin* 563

BIOCHEMISTRY.—MAGNESIUM ANTAGONISM OF THE UNCOUPLING OF OXIDATIVE PHOSPHORYLATION BY IODO-THYRONINES . . . . . *By S. Harvey Mudd, Janey Harting Park, and Fritz Lipmann* 571

BIOCHEMISTRY.—ALLELIC STRAINS OF *Neurospora* LACKING TRYPTOPHAN SYNTHETASE: A PRELIMINARY IMMUNOCHEMICAL CHARACTERIZATION . . . . . *By Sigmund R. Suskind, Charles Yanofsky, and David M. Bonner* 577

MATHEMATICS.—ON ABELIAN VARIETIES OVER FUNCTION FIELDS . . *By Wei-Liang Chow* 582

MATHEMATICS.—THE STRONG LAW OF LARGE NUMBERS WHEN THE FIRST MOMENT DOES NOT EXIST . . . . . *By C. Derman and H. Robbins* 586

MATHEMATICS.—CESÀRO SUMMABILITY OF WALSH-FOURIER SERIES. . . . *By N. J. Fine* 588

MATHEMATICS.—GENERALIZED DERIVATIVES AND EXPANSIONS . . . . . *By Karl Menger and S. S. Shu* 591

MATHEMATICS.—ON THE MODULAR REPRESENTATIONS OF THE SYMMETRIC GROUP VI . . . . . *By G. de B. Robinson and O. E. Taulbee* 596

MATHEMATICS.—THE VALIDITY OF KRON'S METHOD OF TEARING . . . . *By J. P. Roth* 599

MATHEMATICS.—NECESSARY AND SUFFICIENT CONDITIONS FOR CARLSON'S THEOREM ON ENTIRE FUNCTIONS . . . . . *By L. A. Rubel* 601